JUNE 1989

PUBLIC HEALTH AND ENVIRONMENTAL EVALUATION PLAN

REMEDIAL INVESTIGATION / FEASIBILITY STUDY
NAVAL AIR STATION ALAMEDA
ALAMEDA, CALIFORNIA

VOLUME 7

DEPARTMENT OF THE NAVY
WESTERN DIVISION
NAVAL FACILITIES ENGINEERING COMMAND
SAN BRUNO, CALIFORNIA 94066-0727

WORK PLAN VOLUME 7
PUBLIC HEALTH AND ENVIRONMENTAL EVALUATION PLAN
REMEDIAL INVESTIGATION/FEASIBILITY STUDY
NAVAL AIR STATION, ALAMEDA, ALAMEDA, CA

Clement Associates Incorporated
160 Spear Street, Suite 1380
San Francisco, CA 94105-1535 415-957-9429

Environmental and Health Science

June 15, 1989

Ms. Bella Dizon (Code 1813 BD)
Western Division Naval Facilities Engineering Command
900 Commodore Drive
San Bruno CA 94066-0720

Dear Ms. Dizon:

Enclosed are twenty (20) copies of the Work Plan Volume 7 - Public Health and Environmental Evaluation Plan for the Remedial Investigation/Feasibility Study for the Naval Air Station Alameda. Attached to this Work Plan is the Final Preliminary Public Health and Environmental Evaluation (PHEE) developed from the existing data base for this facility. This document has been through Clement Associates final QA/QC review and all responses to comments from the California Department of Health Services have been incorporated with your approval.

Please do not hesitate to call Michael Raybourn or myself, if you have any questions. It has been a pleasure working with you and your staff, and I look forward to having the opportunity to work with you in the future.

Sincerely yours,

Jo Ann Weber

cc: J. Babcock
M. Raybourn

FINAL PRELIMINARY PUBLIC HEALTH AND ENVIRONMENTAL EVALUATION PLAN REMEDIAL INVESTIGATION/FEASIBILITY STUDY (RI/FS) VOLUME 7 OF 8

DATED 01 JUNE 1989

THIS RECORD CONTAINS MULTIPLE VOLUMES WHICH HAVE BEEN ENTERED SEPARATELY

VOLUME 1 OF 8 – FINAL SAMPLING PLAN, RI/FS DATED 2/1/90 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000785**

VOLUME 1A OF 8 – FINAL SAMPLING PLAN, SOLID WASTE ASSESSMENT TEST PROPOSAL ADDENDUM, RI/FS DATED 2/1/89 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000311**

VOLUME 1A OF 8 – FINAL SAMPLING PLAN, SOLID WASTE ASSESSMENT TEST PROPOSAL ADDENDUM, RI/FS DATED 12/1/89 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000789**

VOLUME 1A OF 8 – FINAL SAMPLING PLAN, SOLID WASTE ASSESSMENT TEST PROPOSAL ADDENDUM, RI/FS DATED 2/1/90 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000786**

VOLUME 1B OF 8 – FINAL AIR SAMPLING PLAN, RI/FS DATED 12/1/88 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000275**

VOLUME 2 OF 8 – FINAL HEALTH AND SAFETY PLAN, RI/FS DATED 12/1/88 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000274**

VOLUME 2 OF 8 – REVISED FINAL HEALTH AND SAFETY PLAN, RI/FS DATED 5/1/89 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000351**

VOLUME 2 OF 8 – REVISED FINAL HEALTH AND SAFETY PLAN, RI/FS DATED 11/1/89 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000780**

N00236.000371
ALAMEDA POINT
SSIC NO. 5090.3

VOLUME 3 OF 8 – FINAL QUALITY ASSURANCE PROJECT PLAN – QUALITY ASSURANCE / QUALITY CONTROL PLAN, RI/FS DATED 5/1/89 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000341**

VOLUME 3 OF 8 – FINAL QUALITY ASSURANCE PROJECT PLAN – QUALITY ASSURANCE / QUALITY CONTROL PLAN, RI/FS DATED 1/1/90 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000782**

VOLUME 4 OF 8 – COMMUNITY RELATIONS PLAN, RI/FS DATED 2/15/89 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000301**

VOLUME 5 OF 8 – FINAL PROJECT MANAGEMENT PLAN/SCHEDULE, RI/FS DATED 2/1/89 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000322**

VOLUME 6 OF 8 – DATA MANAGEMENT PLAN, RI/FS DATED 5/1/89 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000361**

VOLUME 8 OF 8 – FINAL FEASIBILITY STUDY PLAN, RI/FS DATED 1/1/90 IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000783**

DRAFT FINAL
REMEDIAL INVESTIGATION/FEASIBILITY STUDY
WORK PLAN ADDENDUM

DATED 29 SEPTEMBER 1993

IS ENTERED IN THE DATABASE AND FILED AT ADMINISTRATIVE RECORD NO. **N00236.000858**

TABLE OF CONTENTS

Attachment
Preliminary Public Health and Environmental Evaluation

1.0 INTRODUCTION

This Public Health and Environmental Evaluation Plan (PHEE Plan) has been prepared on behalf of the Navy by Clement Associates as a subcontractor to Canonie Environmental. The PHEE Plan describes tasks to be performed for the Public Health and Environmental Evaluation (PHEE) at the Naval Air Station (NAS), Alameda, California.

The Work Plan for a Remedial Investigation/Feasibility Study (RI/FS) at NAS Alameda is currently under preparation. This Public Health and Environmental Evaluation Plan, while initiated under the Naval Assessment and Control of Installation Pollutants (NACIP) program purview, has been written to satisfy the Comprehensive Environmental Response, Compensation and Liability Act as amended by the Superfund Amendments and Reauthorization Act of 1986 (CERCLA/SARA) remedial investigation program developed by the U.S. Environmental Protection Agency (EPA). It is consistent with EPA guidance on CERCLA/SARA Remedial Investigation Work Plan development.

The Work Plan for the RI/FS at NAS Alameda consists of the following planning documents:

Volume 1	Sampling Plan
Volume 1A	Sampling Plan, SWAT Proposal Addendum
Volume 1B	Air Sampling Plan
Volume 2	Health and Safety Plan
Volume 3	Quality Assurance Project Plan (QAPP), Quality Assurance/Quality Control Plan (QA/QC)
Volume 4	Community Relations Plan
Volume 5	Project Management Plan/Schedule
Volume 6	Data Management Plan
Volume 7	Public Health and Environmental Evaluation Plan
Volume 8	Feasibility Study Plan

This document is Volume 7 of the Work Plan for the RI/FS.

This plan describes the methodology to be followed in assessing the human health and environmental effects presented by hazardous substances which may be identified in the NAS Alameda Remedial Investigations (RI) by Canonie. In addition, the PHEE Plan describes the methodology which will be employed to develop health-based and environmental performance goals used to evaluate remedial action alternatives in the NAS Alameda Feasibility Studies (FS). The PHEE is, therefore, planned to be one of the interpretive links between the RI and the FS.

1.1 Preliminary Public Health and Environmental Evaluation

Attached to this plan is the preliminary Public Health and Environmental Evaluation (PHEE). The preliminary PHEE provides an assessment of the current potential human and environmental threats posed by the twenty identified study areas at NAS Alameda. The preliminary PHEE was developed using available site history and chemical data collected from various sources; no RI data were available for the preliminary PHEE. The final PHEE will be based solely on data collected in the RI.

1.2 Guidance Documents

No standard format or uniform procedures for preparing a PHEE have yet been established by regulatory agencies or professional societies. However, Clement Associates frequently refers to the available guidance documents in preparation of PHEEs for public and private sector clients. The principal guidance documents which will be followed in preparing PHEEs are:

- EPA (October 1986a), Superfund Public Health Evaluation Manual (SPHEM).
- EPA (June 1985a), Guidance on Feasibility Studies under CERCLA, Chapter 5, "Evaluate Protection of Public Health Requirements."

Other endangerment assessment guidance documents, health risk assessment reports, and scientific references will likely be consulted in the preparation of the PHEE. These documents include:

- State of California, Department of Health Services (May 1986), The California Site Mitigation Decision Tree Manual and updated Applied Action Levels (December 1988).
- EPA (1987a), Superfund Exposure Assessment Manual, Draft, U.S. EPA Office of Emergency and Remedial Response, Washington, D.C. Final draft dated September 22, 1987.
- EPA (1985b), Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessments.
- EPA (1986b), "Guideline for Carcinogen Risk Assessment", Federal Register, September 24, 1986, Vol. 51, No. 185, pp. 33992-34003.
- EPA (1986c), "Guideline for Health Risk Assessment of Chemical Mixtures", Federal Register, September 24, 1986, Vol. 51, No. 185, pp. 34014-34025.
- EPA (1987b). Interim Guidance on ARARs.
- EPA (1988). Integrated Risk Information System (IRIS).

1.3 Regulatory Requirements

This plan and the preliminary PHEE were prepared to respond to the following requirements:

- Federal Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980, as amended by the Superfund Amendments and Reauthorization Act of 1986 (SARA);
- National Oil and Hazardous Substances Pollution Contingency Plan (NCP) of 1985;
- California Department of Health Services (DHS) Remedial Action Order (RAO), Docket No. HSA88/89-051, dated July 1, 1988;
- State hazardous waste control regulations found in the California Code of Regulations, California Health and Safety Code and the Porter-Cologne Water Quality Control Act (California Water Code).

1.4 Remedial Investigation/Feasibility Study

Because of the complexity of NAS Alameda, the unknown number of sites where chemicals might be present, and the different chemicals that might be expected at these sites, the Navy intends to investigate NAS Alameda on a site-by-site basis. Twenty study areas have been identified for inclusion in the Remedial Investigation.

The 20 study areas included in the RI/FS process have been assigned to the following waste stream related groups:

1) Maintenance Area
   - -Building 41

2) Oil and Gasoline Areas
   - -Buildings 459, 547, and 162 (Service Stations)
   - -Building 10 (Power Plant)
   - -Area 97 (AVGAS storage)
   - -Oil Refinery
   - -Fire Training Area

3) Pesticide Storage Areas
   - -Building 114

4) Paint Stripping and Plating Areas
   - -Building 5
   - -Building 360
   - -Building 410
   - -Buildings 400 and 530 (Missile Rework)

5) Test Shop
   - -Building 14

6) Transformer Storage Area
   - -Buildings 301 and 389

7) Waste Storage Areas
   - -Cans C-2 Area
   - -Yard D-13

8) Station Sewer System

9) Marine Environments
   -Seaplane Lagoon
   -Estuary (Oakland Inner Harbor)

10) Landfills
   -1943 - 1956 Disposal Area
   -West Beach Landfill

Based on the results of the RI, the Navy may opt to perform a PHEE for each waste stream group. These groupings will be used to tailor the final PHEE to specific waste stream related contaminants. As data are gathered in the RI, these groupings may change. All waste stream specific PHEEs will then be examined for an overall NAS Alameda assessment. The overall assessment will be made after all of the data anticipated to be collected in the RI has been analyzed.

In summary, the PHEE process will address all of the 20 identified study areas at NAS Alameda. An overall PHEE assessment will be made that combines all individual PHEEs and assesses the entire NAS Alameda facility as one entity.

2.0 OBJECTIVES

The PHEE process is an evaluation and interpretation of the RI data and other information. The PHEE examines the collective demographic, geographic, physical, chemical, ecological, and biological factors and data at a site to describe the extent of the potential or actual exposure and associated risk to a receptor.

The objective of the public health and environmental evaluation process is to assess potential impacts on public health and the environment from actual or potential releases resulting from past waste disposal activities at NAS Alameda. A PHEE provides a baseline site-specific risk assessment that evaluates the site and surrounding area in the absence of remediation.

2.1 Assessment of Remedial Investigation Data

In support of RI activities, an objective of the PHEE is to assess the magnitude and probability of actual or potential public health and environmental risks posed by chemical constituents identified during the RI at NAS Alameda. The following environmental sampling data gathered during the RI will be used to perform the PHEE: air, surface water, groundwater, surface soil, subsurface soil, and sediment samples collected from shoreline areas adjacent to known contaminated areas and the identified marine study areas. These data will be used in the PHEE to assess the no-action remedial alternative.

The details of the environmental sampling program are described in Volumes 1 – Sampling Plan, 1A – Sampling Plan - SWAT Addendum, and 1B – Air Sampling Plan of the Work Plan for NAS Alameda. Data management objectives and protocols are presented in Volume 6 – Data Management Plan.

2.2 Developing Feasibility Study Goals

In support of FS activities, the PHEE develops human health-based and environmental performance goals for evaluating remedial action alternatives in the FS. Public health and environmental concerns are considered in two FS tasks:

- conducting the initial screening of remedial action technologies; and
- preparing a detailed technical evaluation of remedial action alternatives combining technologies that meet the initial screening criteria.

Part of this process will include the identification of applicable or relevant and appropriate requirements (ARARs) using EPA guidance for contaminants selected as indicator chemicals for the site. These ARARs will be compared to on-site concentrations and will be considered in the evaluation of remedial action alternatives.

3.0 PUBLIC HEALTH AND ENVIRONMENTAL EVALUATION PROCESS

Currently, there is no model statement of work for a PHEE as there is for an RI or FS. The PHEE process has however, been generally described and defined in health and environmental risk assessment reports, endangerment assessment guidance documents, and scientific references cited in Section 1.0. These documents were consulted in the preparation of this plan.

The guidance documents cited provide information on the three basic elements of a risk assessment. These are: 1) hazard identification, 2) exposure assessment, and 3) risk characterization. These three elements will constitute the major tasks for the NAS Alameda PHEE.

The following sections include a description of specific PHEE activities and their associated tasks and a brief description of the anticipated format and methodologies that are expected to be used in the PHEE process.

3.1 Organization of the PHEE

The following outline depicts each task and subtask that is expected in the NAS Alameda PHEE process. Each of the PHEE reports will contain essentially the same major tasks though each will be individually tailored to site-specific concerns.

Task 1.0 Background Information (obtained from the RI)
Task 1.1 Site and Surrounding Areas Description
- Task 1.1.1 Site History
- Task 1.1.2 Site Location
- Task 1.1.3 Site Features
- Task 1.1.4 Land Use/Demographics

Task 1.2 Environmental Characteristics
- Task 1.2.1 Geology
- Task 1.2.2 Hydrology

Task 1.2.3 Topography and Surface Drainage

Task 1.2.4 Hydrogeology

Task 1.2.5 Meteorology and Climatic Conditions

Task 2.0 Hazard Identification

Task 2.1 Chemical Characterization

Task 2.1.1 Surface Soil

Task 2.1.2 Subsurface Soil

Task 2.1.3 Groundwater

Task 2.1.4 Surface Water

Task 2.1.5 Air

Task 2.2 Selection of Indicator Chemicals

Task 2.2.1 Selection Criteria

Task 2.3 Environmental Fate and Transport Characteristics of Indicator Chemicals

Task 2.4 Identification of Applicable or Relevant and Appropriate Requirements (ARARs) for Indicator Chemicals

Task 2.5 Intrinsic Toxicological Properties of Indicator Chemicals

Task 2.5.1 Carcinogenicity

Task 2.5.2 Non-Carcinogenic Effects

Task 2.5.2.1 Reproductive Toxicity/Teratogenicity

Task 2.5.2.2 Mutagenicity

Task 2.5.2.3 Acute Toxicity

Task 2.5.2.4 Chronic Toxicity

Task 3.0 Human Exposure Assessment

Task 3.1 Receptor Identification

Task 3.2 Potential Exposure Pathways

Task 3.2.1 Current-Use Conditions

Task 3.2.2 Future-Use Conditions

Task 3.3 Exposure Assessment Models and Calculations

Task 3.3.1 Ambient Air Exposure Pathway

Task 3.3.1.1 Particulates

Task 3.3.1.2 Volatile Emissions

Task 3.3.2 Surface Water Exposure Pathway

Task 3.3.3 Groundwater Exposure Pathway

Task 3.3.4. Surface Soil

Task 3.3.5 Subsurface and Gas Transport

Task 4.0 Quantitative Human Health Risk Characterization

Task 4.1 Comparison to ARARs

Task 4.2 Estimation of Chronic Daily Intake (CDI) Levels

Task 4.3 Potential Current Risks

Task 4.3.1 Current Noncarcinogenic Risks

Task 4.3.2 Current Carcinogenic Risks

Task 4.4 Potential Future Risks

Task 4.4.1 Future Noncarcinogenic Risks

Task 4.4.2 Future Carcinogenic Risks

Task 5.0 Environmental Assessment

Task 5.1 Environmental Characteristics

Task 5.2 Environmental Sampling Results

Task 5.3 Selection of Indicator Chemicals

Task 5.4 Environmental Toxicity

Task 5.5 Potential Environmental Exposure Pathways

Task 5.6 Environmental Risk Characterization

Task 5.6.1 Current-Use Risks

Task 5.6.2 Future-Use Risks

Task 6.0 Conclusions of the PHEE

Task 6.1 Uncertainty Evaluation

Task 7.0 Report Preparation

Task 8.0 FS Support - Health-based and Environmental Performance Goals for the Feasibility Study

3.2 Task Descriptions and Methodologies

The subsequent discussion includes a brief description of the main PHEE tasks and subtasks outlined above and the methodologies that are expected to be used in the process.

Task 1.0 Background Information

The NAS Alameda site background information generated in the RI will be compiled and summarized in the context of a PHEE.

Task 1.1 Site and Surrounding Areas Description

Data for the following topics will be compiled and briefly summarized:

- site history
- site physical features
- current and future land use
- demographics and location of sensitive human receptor population centers
- proximity of ecologically sensitive areas
- proximity to surface water features
- current and future surface water use
- current and future anticipated source of area drinking water supply
- on-site and surrounding land use

Task 1.2 Environmental Characteristics

Environmental characteristics of NAS Alameda which may influence contaminant migration will be discussed. Regional and site-specific information on geology, hydrology, topography and surface drainage, hydrogeology, and meteorology will be presented in the final PHEE. General and site-specific air quality and meteorologic data will be presented to identify potential migration pathways and the location of downwind receptors.

Task 2.0 Hazard Identification

Hazard identification characterizes the site contaminants with respect to their presence on site, their mobility in the environment, and the available health effect data. Environmental sampling data are compiled and indicator chemicals selected for each media.

Task 2.1 Chemical Characterization

Initially, chemical analytical data, such as soil, groundwater, surface water, and air analyses are examined and characterized in terms of contaminants detected. Detected chemicals will be summarized according to their corresponding geometric mean and maximum concentrations and their frequency of detection. The site will be characterized in terms of the contaminant distribution in each environmental medium: soil, groundwater, surface water, and air. The chemical concentrations will then be compared to available site-specific data on background levels and/or published regional values. The chemical analytical data base will be reviewed for scope, quality, and validity as described in the Quality Assurance Project Plan (Volume 4 of this Work Plan).

Task 2.2 Selection of Indicator Chemicals

Because of the large number of chemicals typically detected at a site, a subset of key chemical contaminants is generally selected in order to focus the PHEE on the chemicals most likely to pose some risk. These "indicator chemicals" are chemicals identified as being site-related and represent the more toxic, mobile, and persistent chemicals at a site, as well as those present in highest concentrations.

Task 2.2.1 Selection Criteria

In selecting indicator chemicals, efforts are made to eliminate those contaminants which are not believed to be site-related. The following

criteria are generally used to remove non-site related contaminants from consideration.

- Comparison With Blanks. If chemicals are detected in travel blanks or field blanks, concentrations of compounds in the site samples and blanks are compared. For those chemicals that are common artifacts of field or laboratory procedures (e.g. acetone, chloroform, phthalate esters, and methylene chloride), concentrations that exceeded field blank concentrations by ten times (10x) are considered to be site-related. Chemicals not meeting these criteria are eliminated from consideration.

- Comparison With Background Concentrations. Concentrations of contaminants detected at the site are compared to concentrations detected in upgradient from the site or regional background levels for inorganics available in the literature. Typically, chemicals with mean concentrations less than twice (2x) background concentrations may be eliminated from consideration.

- Frequency of Detection. When a sufficient number of samples have been collected to ascertain that the site has been well characterized with respect to any individual chemical, the frequency of detection for that chemical may be considered in selecting chemicals of concern. Typically, when at least twenty samples are available, chemicals detected in less than 5% of the samples are not considered to be of concern at the site.

- Historical Use, Concentration, Toxicity, and Physicochemical Properties. Selection of chemicals of concern may also include consideration of the known use of a compound at the site, magnitude of detected concentrations, known toxic potential, and migration characteristics in environmental media. For example, chemicals not known to have been used at the site, that were not found in the leachate, or that are clearly not of toxicological concern may be removed from further consideration even if they satisfy certain of the other criteria outlined above.

The subsequent risk characterization will focus only on these selected indicator chemicals.

Task 2.4 Identification of Applicable or Relevant and Appropriate Requirements (ARARs)

According to the guidelines for preparing risk assessments as part of the RI/FS process (EPA, 1986a; PRC, 1985; CDHS, 1988), the potential adverse

effects on human health should be assessed where possible by comparing chemical concentrations found at or near the site with applicable or relevant and appropriate requirements (ARARs) that have been developed for the protection of human health or the environment. If suitable ARARs are not available for all of the chemicals of concern and for all of the exposure scenarios considered, a quantitative risk evaluation must be completed.

Remedial actions selected under the Superfund Amendments and Reauthorization Act of 1986 (SARA) must attain levels of cleanup of hazardous substances released into the environment and of control of further release which assure protection of human health and the environment. SARA specifies that any selected remedial action must achieve a level of control which at least attains requirements that are legally applicable to the hazardous substances of concern or relevant and appropriate under the circumstances of release or threatened release. Accordingly, EPA guidelines for preparing risk assessments as part of the RI/FS process (EPA, 1986a) recommend comparison of chemical concentrations found at or near a site with ARARs. The California Department of Health Services currently uses EPA's guidance on ARARs (CDHS, 1988). The EPA's interim guidance on ARARs (EPA, 1987b) defines ARARs as follows:

- Applicable Requirements means those cleanup standards, standards of control, and other substantive environmental protection requirements, criteria, or limitations promulgated under Federal or State law that specifically address a hazardous substance, pollutant, contaminant, remedial action, location, or other circumstance at a CERCLA site. "Applicability" implies that the remedial action or the circumstances at the site satisfy all of the jurisdictional prerequisites of a requirement....

- Relevant and Appropriate Requirements means those cleanup standards, standards of control, and other substantive environmental protection requirements, criteria, or limitations promulgated under Federal or State law that, while not "applicable" to a hazardous substance, pollutant, contaminant, remedial action, location, or other circumstance at a CERCLA site, address problems or situations sufficiently similar to those encountered at the CERCLA site that their use is well suited to the particular site.

> The relevance and appropriateness of a requirement can be judged by comparing a number of factors, including the characteristics of the remedial action, the hazardous substances in question, or the physical circumstances of the site, with those addressed in the requirement. It is also helpful to look at the objective and origin of the requirement. For example, while RCRA regulations are not applicable to closing undisturbed hazardous waste in place, the RCRA regulation for closure by capping may be deemed relevant and appropriate.
>
> A requirement that is judged to be relevant and appropriate must be complied with to the same degree as if it were applicable. However, there is more discretion in this determination: it is possible for only part of a requirement to be considered relevant and appropriate, the rest being dismissed if judged not to be relevant and appropriate in a given case.
>
> Non-promulgated advisories or guidance documents issued by Federal or State governments do not have the status of potential ARARs. However, ..., they may be considered in determining the necessary level of cleanup for protection of health or environment.

Only those ARARs or advisories or guidance that are ambient or chemical-specific requirements (i.e., those requirements which "set health or risk-based concentration limits or ranges in various environmental media for specific hazardous substances, pollutants, or contaminants") as opposed to ARARs which are classified as action-specific or locational are used in risk assessment (EPA, 1987). Under SARA, EPA at a minimum currently considers maximum contaminant levels (MCLs) developed under the Safe Drinking Water Act (SWDA), national ambient air quality standards (NAAQS) promulgated under the Clean Air Act, and state drinking water and ambient air standards to be potential ARARs for use in risk assessment at Superfund sites. CDHS also considers National Emission Standards for Hazardous Air Pollutants (NESHAPs) as ARARs. In addition, other relevant criteria or guidance (eg., maximum contaminant level goals (MCLGs) promulgated under the SDWA, EPA Ambient Water Quality Criteria (AWQC) and California Applied Action Levels) may be useful in assessing baseline risks or developing goals for remedial action.

Task 2.5 Intrinsic Toxicological Properties of Indicator Chemicals

The available toxicological literature for the indicator chemicals will be collected and summarized. The most recent toxicological data on cancer potency factors and reference doses (RfDs) for indicator chemicals will be identified using EPA's Integrated Risk Information System (IRIS) and other current toxicological information.

The human health and environmental hazards to be evaluated in the PHEE will include but are not limited to:

- carcinogenicity;
- reproductive toxicity;
- teratogenicity (production of malformed offspring);
- genotoxicity (genetic toxicity);
- acute toxicity (refers to the effects that result from very short-term, usually single dose, exposure to material); and
- chronic toxicity (refers to effects that occur after long-term exposure, i.e., a significant portion of the animal's lifespan).

Task 3.0 Human Exposure Assessment

As part of the PHEE, all known or potential exposure pathways associated with the identified receptors are assessed to determine their significance. Exposure pathways associated with the site will be examined and evaluated for completeness. Potentially important pathways will be identified for the subsequent, more detailed, characterization of risk.

For an actual exposure to occur, or a potential exposure to be viable, there must be a complete exposure pathway. In order for a chemical to pose a human health risk, a complete exposure pathway must be identified. A complete exposure pathway consists of four elements: 1) a source and mechanism of chemical release to the environment, 2) an environmental transport medium (eg., air or soil) for the released chemical, 3) a point of potential human contact with the contaminated medium (the exposure point), and 4) a human exposure route (eg., inhalation) at the contact point (EPA, 1986a). The possible pathways of human exposure to constituents from the NAS Alameda site

under existing and future conditions will be examined in the final PHEE. These pathways will consider potential releases from the site into soil, ground water, surface water, and air.

The attached preliminary PHEE assesses the base of potential site exposure pathways. These include: airborne exposure pathways, potential exposure due to direct contact with surface soil, and off-site migration of groundwater and surface water runoff with subsequent potential exposure to environmental receptors.

Task 3.1 Receptor Identification

Various types of data will be collected in order to identify potential receptors in the site vicinity.

Demographic Data: Demographic data gathered during the RI will be summarized with respect to population clusters and potentially sensitive subpopulations (eg., children or elderly adults). Information on the number of females and males residing in the area will be noted since there may be sexual differences in susceptibility to various health effects.

Water Well Inventory: As part of the RI, domestic and irrigation water wells on and adjacent to NAS Alameda will be identified. Information gained through the RI will be used to determine potential interconnectedness of aquifers and possible groundwater receptors.

Surface Water Users: The proximity of surface water bodies and their use for drinking water and recreational purposes (e.g., fishing, boating, and swimming) will be examined.

Task 3.2 Potential Receptors and Exposure Pathways

Possible exposure pathways and circumstances in which exposure may occur are identified. Exposure is considered via the conventional modes:

inhalation, ingestion, and dermal exposure. The time periods evaluated are current conditions and future "no action" conditions (i.e., no remediation is performed).

Human receptors are categorized in the context of possible exposure as workers both on and off site (occupational exposure), residents living in the immediate vicinity of the site, or residents on-site in the future, or recreational users of surface water. Determination of exposure points is site-specific and the possibility of other exposure points may exist currently or in the future. This possibility will be examined and the completeness of the pathway to a suitable receptor will be judged.

Potentially complete present and future exposure pathways and receptor points will be presented and assessed. It may be determined that some of these potentially complete exposure pathways represent hypothetical or insignificant exposures to receptors. However, the purpose of the pathway assessment is to determine which of these pathways might be complete and has the potential to result in toxicologically significant human exposure.

Task 3.3 Exposure Assessment Models and Calculations

For an in-depth quantitative assessment of environmental fate and transport, modeling techniques will be employed. This PHEE Plan describes the types of models and calculations which will be examined for applicability. It does not specify exactly which models or types of calculations will be used in the exposure assessment; the choice of model will be made after reviewing site RI data.

The selection of a model used in the PHEE and the development of the input data will carefully consider site-specific conditions. The site-specific approach first requires review of the collected RI data. The complexity of the site conditions and the extent of the data base for the site control the applicability of a given model. The model selected must be able to reasonably simulate the environmental conditions in the area of interest

and the constituent transport processes. In order to be useful, models used in the PHEE should result in output that is consistent with the available data (i.e., the model should be capable of being calibrated).

Computer modeling procedures usually must be applied to quantify atmospheric fate, surface water fate, and groundwater fate.

Task 3.3.1 Ambient Air Exposure

In situations where air data are incomplete and soil sampling results are available, airborne emissions of contaminants may be estimated using a mathematical model. Releases via dispersion of airborne particulate, passive diffusion from soils, and subsurface vapor migration can be estimated using these models.

Generally, all air dispersion models require a chemical emission rate in order to estimate the concentration of the constituent of concern at some distant location. The methods for estimating airborne particulate (fugitive dust) emission rates area detailed in Cowherd (1985) and the Superfund Exposure Assessment Manual (EPA, 1987a). Organic vapor emission rates can be estimated for landfill-type situations with and without internal gas generation under a varying set of conditions using methods discussed in Karimi et al. (1987).

Thus, chemical emission rates can be calculated using the appropriate model for site-specific circumstances. Once the receptor location and the ambient air concentrations of the indicator chemicals are estimated, those values will be used to characterize the risks associated with air releases from the site.

In addition to modeling potential volatile and particulate emissions, actual ambient air data will be collected. The draft Work Plan Volume 1B, Air Sampling Plan, (Canonie/WESTDIV, 1988c) describes the NAS Alameda air sampling

program. In addition, site meteorological conditions will be examined to evaluate air dispersion characteristics at the site.

Task 3.3.2 Surface Water Exposure Pathway

In order to evaluate potential surface water pathways (i.e., consumption of contaminated fish), it may be necessary to estimate the rate of contaminant uptake. Values from available scientific literature will be used to predict the rate of uptake of contaminants by fish.

Task 3.3.3 Groundwater Exposure Pathway

In the groundwater exposure pathway assessment, a variety of groundwater models may be employed to generate exposure point concentrations. Since groundwater data are being collected during the RI, this data may be extrapolated to predict concentrations outside the study area.

Subsurface soil contaminants may be modeled to predict the rate of leaching from soils to groundwater. Models from the California DHS Exposure Assessment Manual, the Superfund Exposure Assessment Manual, or the available literature may be employed to estimate potential groundwater concentrations of subsurface soil contaminants. When the RI data is available, an appropriate model will be selected.

Task 4.0 Quantitative Human Health Risk Characterization

The objective of the risk characterization will be to estimate the incidence of an adverse health effect under the various conditions of exposure defined in the exposure assessment. It will be performed by integrating information developed during the exposure and hazard assessments to yield a complete characterization of risk at the site.

The initial phase of the risk characterization will be the comparison of concentrations of site contaminants to the identified ARARs. For those

chemicals or media where ARARs do not exist, a quantitative risk characterization will be performed.

To quantitatively assess the potential risks to human health associated with the current-use and future-use exposure scenarios considered in this assessment, the concentrations of chemicals in relevant environmental media at points of potential exposure (exposure point concentrations) are converted to chronic daily intakes (CDIs). CDIs are expressed as the amount of a substance taken into the body per unit body weight per unit time, or mg/kg/day. A CDI is averaged over a lifetime for carcinogens (EPA, 1986b) and over the exposure period for noncarcinogens (EPA, 1986c). For potential carcinogens, excess lifetime cancer risks are obtained by multiplying the daily intake of the contaminant under consideration by its cancer potency factor.

Potential risks for noncarcinogens are presented as the ratio of the chronic daily intake exposure to the reference dose (CDI:RfD). The sum of all of the ratios of chemicals under consideration is called the hazard index. The hazard index is useful as a reference point for gauging the potential effects of environmental exposures to complex mixtures. In general, hazard indices which are less than one are not likely to be associated with any health risks, and are therefore less likely to be of regulatory concern than hazard indices greater than one. A conclusion should not be categorically drawn, however, that all hazard indices less than one are "acceptable" or that hazard indices of greater than one are "unacceptable". This is a consequence of the perhaps one order of magnitude or greater uncertainty inherent in estimates of the RfD and CDI in addition to the fact that the uncertainties associated with the individual terms in the hazard index calculation are additive.

In accordance with EPA's guidelines for evaluating the potential toxicity of complex mixtures (EPA, 1986d), it was assumed that the toxic effects of the site-related chemicals would be additive. Thus, lifetime excess cancer risks and the CDI:RfD ratios were summed to indicate the potential risks associated with mixtures of potential carcinogens and

noncarcinogens, respectively. In the absence of specific information on the toxicity of the mixture to be assessed or on similar mixtures, EPA guidelines generally recommend assuming that the effects of different components on the mixtures are additive when affecting a particular organ or system. Synergistic or antagonistic interactions may be taken into account if there is specific information on particular combinations of chemicals.

Task 5.0 Environmental Assessment

In this section of the PHEE, potential impacts to non-human receptors are evaluated. The steps for this environmental assessment roughly parallel those for the human risk assessment, in that information on exposure and toxicity are combined to generate an estimate of impact. However, the goal of human health risk assessment is protection of the individual. While protection of individual wild organisms also may be important (e.g., the death of one individual organism of an endangered species), in most cases environmental risk assessment is focused at the population level (e.g., decreased salmon biomass). In many cases, there is a paucity of toxicity data relevant to wildlife and it is difficult to draw inferences to the population level. For these reasons, environmental risk assessments must to a large degree be qualitative.

Task 5.1 Environmental Characteristics

Areas where releases of hazardous substances may be capable of doing harm to non-human receptors will be identified. These locations may include national or state parks, habitats of threatened or endangered species, national wildlife refuges, fish and wildlife management areas, wetlands used as breeding grounds, estuaries where spawning or breeding takes place, and commercial fishing or shell fish harvesting areas. Potential non-human receptors will also be identified. These may include known benthic populations such as shellfish beds, in the vicinity of NAS Alameda which could be impacted by chemicals in the surface water. State and local environmental agencies will be contacted for baseline information on wildlife distribution.

Available public information documents such as environmental impact statements will be consulted.

Task 5.2 Environmental Sampling Results

Sampling results collected from media pertinent to the environmental assessment (i.e., biota) will be summarized and discussed in comparison to other relevant data (i.e., surface water and sediments).

Task 5.3 Selection of Indicator Chemicals

As in human health assessment, an effort is made to focus on contaminants which contribute to environmental risk. These indicator chemicals are identified as being site-related and representing the more toxic, mobile, and environmentally persistent chemicals present.

Task 5.4 Environmental Toxicity

In this section, a brief description of the toxic effects of site-related chemicals to biota will be provided. In addition, any available chemical-specific standards, criteria, and guidance will be identified. Federal Ambient Water Quality Criteria (AWQC) exist for protection of freshwater and marine aquatic life; however, no such values have been developed for the protection of terrestrial life.

Task 5.6 Environmental Risk Characterization

Site-specific exposure levels and estimated environmental concentrations will be compared to ecotoxicity data and to existing environmental concern levels such as potential for bioconcentration in shellfish and regulatory guidelines and standards. The uncertainties will be characterized.

Task 6.0 Conclusions of the PHEE

The PHEE will evaluate the available data to formulate a conclusion on NAS Alameda's potential impact on human health and the environment currently or in the future. Assumptions made during quantification and uncertainties inherent in the risk assessment process will be identified.

Task 7.0 Report Preparation

The culmination of the PHEE process will be the generation of a report which will address all aspects of the PHEE process. If site-specific PHEEs are prepared, a final summary PHEE report will be created that reviews data from the individual PHEEs and addresses the entire NAS Alameda facility. All PHEE reports will be submitted to the California Department of Health Services.

4.0 REFERENCES

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (CDHS). 1986. The California Site Migitation Decision Tree.

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (CDHS). 1988. Personal Communication with John Christopher.

CANONIE ENVIRONMENTAL SERVICES CORP./WESTDIV. (Canonie/WESTDIV). 1988a. "Air Sampling Plan, Remedial Investigation/Feasibility Study, Naval Air Station Alameda, Alameda, California, Volume 1.B," prepared for Department of the Navy, Western Division, Naval Facilities Engineering Command, August.

CANONIE ENVIRONMENTAL SERVICES CORP./WESTDIV. (Canonie/WESTDIV). 1988b. "Quality Assurance Project Plan (QAPP), Quality Assurance/Quality Control Plan (QA/QC), Remedial Investigation/Feasibility Study, Naval Air Station Alameda, Alameda, California, Volume 3," prepared for Department of the Navy, Western Division, Naval Facilities Engineering Command, August.

CANONIE ENVIRONMENTAL SERVICES, CORP./WESTDIV. (Canonie/WESTDIV). 1988c. "Draft Sampling Plan, Remedial Investigation/Feasibility Study, Naval Air Station Alameda, Alameda, California, Volume 1a," prepared for Department of Navy, Western Division, Naval Facilities Engineering Command, October.

COWHERD, et al. 1985. Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites, Kansas City, MO., Midwest Research Institute. PB85-192219

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Guidance on Feasibility Studies under CERCLA, Chapter 5, "Evaluate Protection of Public Health Requirements", Office of Research Development. June 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessments. Office of Health and Environmental Assessment. OHEA-E-161. March 1985

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Superfund Public Health Evaluation Manual. EPA 540/1-86-060. October 1986

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Guidelines for Carcinogenic Risk Assessment. Fed. Reg. 51:33992-24003

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986c. Guidelines for Health Risk Assessment of Chemical Mixtures. Federal Register. September 24, 1986

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Superfund Exposure Assessment Manual. Final Draft. Office of Emergency and Remedial Response. September 1987

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. Superfund Program: Interim Guidance on Compliance with Applicable or Relevant and Appropriate Requirements. Fed. Reg. 52:32496-32499

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, Ohio

ATTACHMENT

PRELIMINARY PUBLIC HEALTH AND ENVIRONMENTAL EVALUATION

TABLE OF CONTENTS

LIST OF TABLES

LIST OF TABLES (cont.)

LIST OF FIGURES

1.0 INTRODUCTION

This report constitutes a preliminary assessment of health risks for the 20 identified sites on the Naval Air Station (NAS) Alameda in Alameda, California, as currently identified in the Remedial Investigation/Feasibility Study being conducted by Canonie Environmental Services Corporation (Canonie). This introduction for the preliminary Public Health and Environmental Evaluation (PHEE) contains information on the report authorization, objectives, organization and limitations.

1.1 AUTHORIZATION

The Work Plan for a Remedial Investigation/Feasibility Study (RI/FS) at NAS Alameda is currently under preparation. This preliminary PHEE, while initiated under the purview of the Naval Assessment and Control of Installation Pollutants (NACIP) program purview, has been written to satisfy the Comprehensive Environmental Response, Compensation and Liability Act, as amended by the Superfund Amendments and Reauthorization Act of 1986 (CERCLA/SARA) remedial investigation program developed by the U.S. Environmental Protection Agency (EPA). This preliminary PHEE was written to respond to the Remedial Action Order (RAO), Docket No. HSA 88/89 - 051 from the California Department of Health Services. It is consistent with EPA guidance on CERCLA/SARA Remedial Investigation Work Plan development.

The Work Plan for the RI/FS at NAS Alameda consists of the following planning documents:

Volume 1	Sampling Plan
Volume 1A	Sampling Plan, SWAT Proposal Addendum
Volume 1B	Air Sampling Plan
Volume 2	Health and Safety Plan
Volume 3	Quality Assurance Project Plan (QAPP), Quality Assurance/Quality Control Plan (QA/QC)
Volume 4	Community Relations Plan
Volume 5	Project Management Plan/Schedule

Volume 6 Data Management Plan
Volume 7 Public Health and Environmental Evaluation Plan
Volume 8 Feasibility Study Plan

1.2 OBJECTIVES

A public health and environmental evaluation is prepared as part of the evaluation of a site subject to cleanup action under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) of 1980, as amended in 1986 under the Superfund Amendments and Reauthorization Act (SARA), and under the State of California Hazardous Substance Cleanup Act of 1984. A PHEE is performed to determine if an actual or threatened release of a hazardous substance from the site may pose a risk to public health or the environment. This preliminary PHEE and data gaps assessment is designed to be used as a scoping tool in order to direct RI sampling efforts to the data requirements of the final PHEE. The preliminary PHEE characterizes the physical description of the site and identifies contaminants detected or suspected to be present at the site. The toxic properties of the identified contaminants are discussed and exposure pathways of potential concern are identified. Finally, data gaps are identified in order to focus RI sampling efforts to generate data necessary for the final quantitative PHEE. Upon evaluation of the RI data, some of the exposure pathways and chemicals of concern discussed in the preliminary PHEE report may change significantly in the final PHEE.

1.3 ORGANIZATION

In Section 2 of this preliminary PHEE, the 20 identified sites on the NAS Alameda are described with respect to their history, use and waste management practices. Additionally, Section 2 presents information on overall site environmental characteristics and presents available data on site contamination. Section 3 presents information on the toxicity and regulatory standards of the chemicals selected as potential chemicals of concern. Section 4 identifies human exposure pathways and discusses the environmental

fate and transport of the chemicals identified as potential chemicals of concern. Section 5 presents a qualitative human health risk characterization; Section 6 qualitatively assesses the risks to the environment. Data needs for the final PHEE are presented in Section 7. The conclusions of the preliminary PHEE are discussed in Section 8. References for this report are included in Section 9.

1.4 LIMITATIONS

The data available for inclusion in this preliminary PHEE are very limited. Although waste handling practices at NAS Alameda are well documented, little information is available on environmental contamination. This document presents a preliminary (level I) PHEE for NAS Alameda. As a Level I PHEE, this assessment is qualitative in nature and limited in scope to soil, groundwater, surface water and sediment data collected prior to the initiation of the RI.

2.0 SITE CHARACTERISTICS

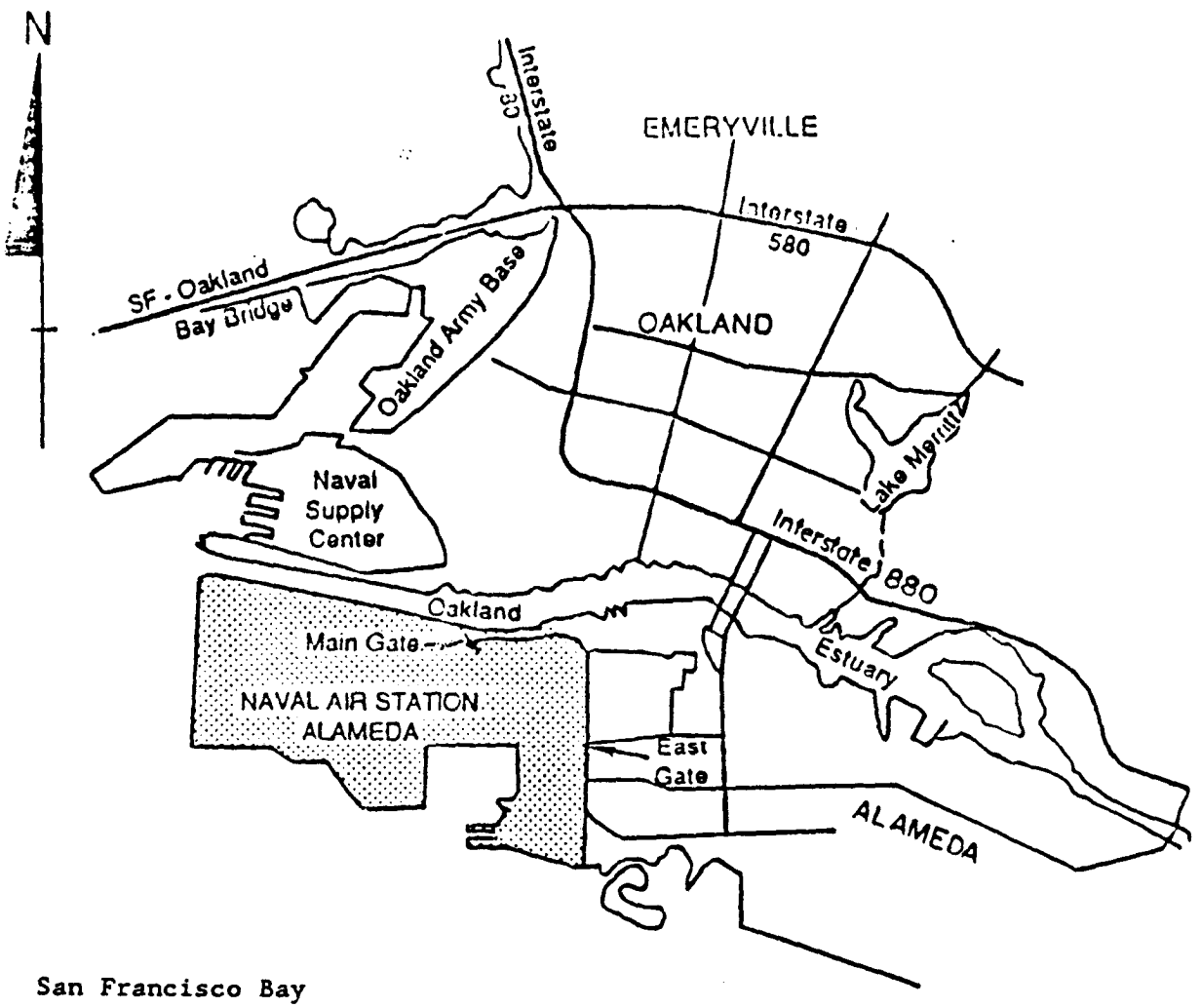
2.1 SITE DESCRIPTIONS

The Naval Air Station Alameda (NAS Alameda) occupies the western tip of the island of Alameda and is located in Alameda and San Francisco Counties, California. Alameda Island is found along the eastern side of San Francisco Bay, as shown on the location map presented in Figure 2-1. Alameda NAS occupies approximately 2,634 acres. Most of the eastern portion of the station is developed with offices and industrial facilities. Runways and support facilities occupy the western part of the station (Canonie/WESTDIV, 1988a).

On the basis of previous investigations at the site, a total of 20 separate study areas have been identified as being areas of potential concern (Alliance/WESTDIV, 1987). The locations of the study areas are presented in Figure 2-2. Each study area is described below. A general description of the area is presented, along with a discussion of the industrial processes reported to have occurred in the area and the wastes generated due to these activities. Chemical data from previous sampling investigations are presented by study area in Section 2.3.

2.1.1 Building 41 (Maintenance Area)

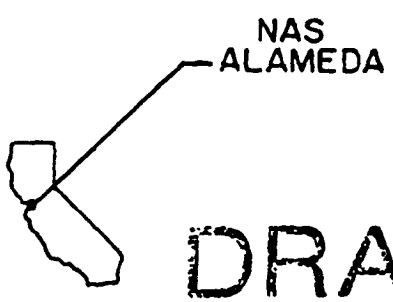
Building 41, formerly used as a hangar for seaplanes, is one of several hangars located along the northern boundary of the Seaplane Lagoon. Aerial photographs indicate that Building 41 was constructed and the surrounding area paved in the early 1940s (Canonie/WESTDIV, 1988b). Aircraft maintenance is currently conducted in Building 41. Primary activities involve the intermediate repair of aircraft components for transient and resident aircraft. Aircraft subsystems such as hydraulics, brakes, avionics, engines, electrical wiring, and instrumentation are maintained and repaired. Calibration of testing equipment is also performed. Aircraft maintenance and repair activities consist of paint stripping, painting, sandblasting,



SITE LOCATION

SCALE

6000 0 6000 FEET



DRAFT

SITE LOCATION PLAN
NAVAL AIR STATION
ALAMEDA, CALIFORNIA

PREPARED FOR

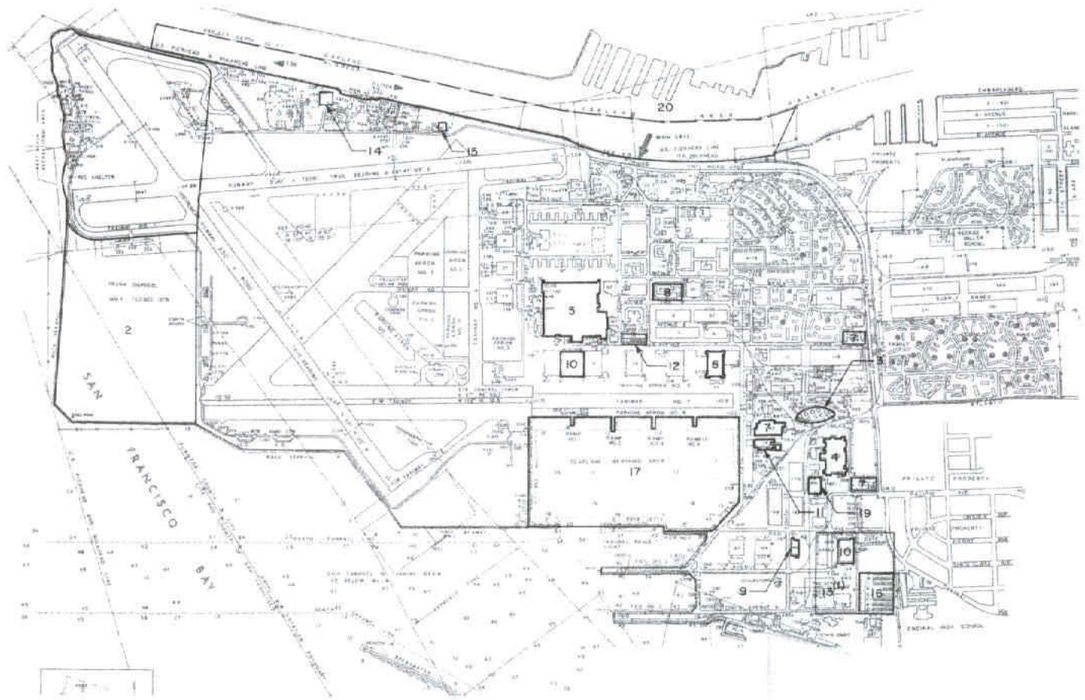
WESTERN DIVISION
NAVAL FACILITIES ENGINEERING
COMMAND

Canonie Environmental

DATE: 5-4-88	FIGURE 2-1	DRAWING NUMBER 86-018-A1
SCALE: AS SHOWN		

REVISIONS	NO.	DATE

DRAWN BY: LEH 5-4-88 | CHECKED BY | APPROVED BY | DRAWING NUMBER 86-018-A1



NOTES:

1. THE AREA OF SITE 13 INCLUDES THE AREAS OF SITES 10 AND 16.

LEGEND:

Site No.	Site Description
1	1943-1956 Disposal Site
2	West Beach Landfill
3	Area 97 (Aviation Gasoline Tanks)
4	Building 360 (Plating Shop, Engine Cleaning Shop, Paint Shop, and Paint Stripping Shop)
5	Building 5 (Plating Shop, Paint Stripping Shop, Cleaning Shop, and Paint Shop)
6	Building 41 (Aircraft Intermediate Maintenance Dept.)
7	Buildings 162, 459, and 543 (Service Stations)
8	Building 114 (Pest Control Area and Separator Pit)
9	Building 410 (Paint Stripping)
10	Buildings 400 and 530 (Missile Rework Operations)
11	Building 14 (Engine Test Cell)
12	Building 10 (Power Plant)
13	Oil Refinery
14	Fire Training Area
15	Buildings 301 and 389 (Storage Area)
16	Cans C-2 Area
17	Seaplane Lagoon
18	Station Sewer System (Not on Site)
19	Yard D-13 (Hazardous Waste Solvents)
20	Estuary (Oakland Inner Harbor)

SCALE
1000 0 1000 2000 FEET

REMEDIAL INVESTIGATION/FEASIBILITY
STUDY SITES
NAVAL AIR STATION
ALAMEDA, CALIFORNIA

PREPARED FOR

US NAVY
ALAMEDA, CALIFORNIA

CanonieEnvironmental

DATE: 7-27-88	FIGURE 1-2	DRAWING NUMBER 86-018-E3
SCALE:		86-018-E2

REVISIONS	NO.	DATE

DRAWN BY	CHECKED BY	DRAWING NUMBER
LEA 7-22-88	LEA 7-27-88	86-018-E3

degreasing, washing, repairing hydraulic systems, brakes, electrical wiring, and overhauling engines and associated gear.

Canonie/WESTDIV (1988a) described current conditions at Building 41 based on a site visit in March 1988. A paint stripping tank (3' x 5' x 1') located adjacent to the west side of Building 41 is used for stripping small parts. Rinse waters are discharged to East Bay Municipal Utility District through a nearby sewer manhole. A storm drain is located adjacent to the paint stripping tank. Although visible signs of contamination in the storm drain from overflows of the stripping tank were not observed in March 1988, past waste disposal practices suggest that some stripping wastes have spilled into the storm drain which empties into the Seaplane Lagoon (ERM-West/Aqua Resources/WESTDIV, 1987). Approximately thirty (30) 55-gallon drums containing a variety of wastes are currently stored west of Building 41. This area is apparently a temporary staging area for the off-site disposal of hazardous wastes.

Wastes stored or removed from Building 41 include: PD 680 dry cleaner, trichlorotrifluoroethane solvent, 6083 oil, trichloroethane solvent, paint wastes and strippers, lead-acid type batteries, and used hydraulic fluid. Up to 100 drums of wastes are reported to have been stored at an unreported location outside of Building 41 (Alliance/WESTDIV, 1987). Two 300-gallon, above ground metal containers (bowsers) were used to store hydrocarbon wastes prior to off-site disposal. The containers were located outside of Building 41 at the northeast and northwest corners (ERM-West/Aqua Resources/WESTDIV, 1987). No documented releases of wastes are recorded. Any surface spills would probably flow into storm drains because most of the area surrounding Building 41 is concrete or paved. However, chemicals from unreported spills may have migrated through cracks in the pavement into the underlying soil.

2.1.2 Buildings 162, 459, and 547 (Service Stations)

Three service stations are located or were formerly located at Building 162, 459, and 547. Two of these location (Buildings 459 and 547) are the site

of underground gasoline or waste oil tanks that are currently leaking and that have been known to leak in the past (E & E/WESTDIV, 1983; Canonie/WESTDIV, 1988a).

Building 162: Canonie/WESTDIV (1988a) reported that no information has been located that indicates the presence of underground storage tanks on the site currently occupied by Building 162. A Navy Exchange, which commonly includes a service station, formerly operated on the site. Currently, several operations are housed in Building 162: the Fuel Systems Accessories Shop, the Pump and Controls Shop, and the Regulator and Compressor Shop.

Current wastes from the operations in Building 162 include small quantities of waste solvents such trichloroethane (PD-680), mixed lube and hydraulic oils, metal shavings, acetone, and freon. These wastes are collected in drums and stored in Building 112 prior to off-base disposal (ERM-West/Aqua Resources/WESTDIV, 1987).

Building 459: Building 459 has been in continuous use from 1964 as a service station. Gasoline is currently stored in three underground 10,000-gallon stainless steel tanks. These three tanks were reported to have plumbing leaks in a recent tank testing study (ERM-West/Aqua Resources/WESTDIV, 1987; as cited in Canonie/WESTDIV, 1988a). A fourth tank was used in the past; however, due to suspected leaks it has been taken out of service. Waste oil is stored in an underground steel tank located adjacent to an auto shop on the western side of Building 459. In the past, three underground gas tanks were abandoned in place due to suspected leaks (ERM-West/Aqua Resources/WESTDIV, 1987).

In 1982, fuel lines from underground gasoline tanks to the gas pumps were found to be leaking. The lines were dug up and replaced. Visible oil sheen was reported in the trenches during excavation. Contents of the waste oil tank located at Building 459 are known to have periodically backed up into the auto shop (Alliance/WESTDIV, 1987).

Waste oils which are stored in the waste oil tank and are pumped out and disposed of off site are the only wastes reported to be generated at Building 459 (ERM-West/Aqua Resources/WESTDIV, 1987)

Building 547: Building 547 is a base annex service station that is also accessible from outside the base. It was constructed in 1971 and was in operation until approximately 1980. Three 12,000-gallon underground fiberglass tanks were installed at the time of construction. In addition, two stainless steel underground tanks located on the northwestern corner of Building 547 are used to store waste oil. One tank has a capacity of 5,000-gallons, and the other a 10,000-gallon capacity (Alliance/WESTDIV, 1987).

In 1980, one of the fiberglass tanks ruptured and was taken out of service. The remaining two of the gasoline tanks were reported to have plumbing leaks in a recent tank testing survey (ERM-West/1987; as cited in Canonie/WESTDIV, 1988a).

2.1.3 Building 10 (Power Plant)

Building 10 is the site of a steam generation power plant and was built in the early 1940s with seven boilers. Building 10 is located one street north of the Seaplane Lagoon near Building 5. Natural gas is the primary fuel source and diesel fuel is the back-up fuel. Eight aboveground tanks with a total capacity of 150,000 gallons were installed and bermed on the south side of the plant and are currently in use. A recent tank testing study discovered five underground tanks at Building 10. Four were found to be filled with sand and the fifth was proposed for removal (Canonie/WESTDIV, 1988a).

Bunker C fuel was originally used until the early 1970s and was stored in eight underground storage tanks on the north side of the building which are now reported as abandoned. It is not known if three of these tanks were removed at some time in the past or not discovered in the recent tank survey. Seven of the underground storage tanks each had a capacity of 12,000 gallons and one of the underground storage tanks had a 24,000-gallon capacity. Bunker

C fuel oil spills have occurred in the past and fuel has accumulated in the steam pipe trenches north of the building. Suction trucks usually skimmed oil off the surface and disposed of it in the oil sump at West Beach Landfill. No spills have been reported after the early 1970s. The possibility for surface and subsurface soil and groundwater contamination exists due to earlier spills and leaks (E & E/WESTDIV, 1983).

Bunker C fuel oil used at NAS Alameda was probably a mixture of petroleum residues and cutter stocks (e.g., light cycle oils, diesel or jet fuel). This material may have contained significant amounts of polycyclic aromatic hydrocarbons (PAHs). Monocyclic aromatics (e.g., benzene, toluene, and xylene) may have been present in formulations containing jet fuel (Chevron, 1985).

Current waste generated in Building 10 includes boiler blowdown containing caustic soda, phosphate, and sulfide; the waste is discharged to the sanitary sewer system. Two 600-gallon bowsers (metal storage containers) are maintained for chemicals (morpholine) and for boiler water treatment and waste oil (ERM-West/Aqua Resources/WESTDIV, 1987).

2.1.4 Area 97

Area 97 is located immediately west of the East Gate of the NAS Alameda. Five partially buried tanks which stored aviation gasoline (AVGAS) were formerly located in a 2-acre parcel within Area 97 (Canonie/WESTDIV, 1988a). Currently, a well-maintained lawn with an installed sprinkler system was observed at the former location of the tanks in Area 97 on a site visit in September 1988 by Clement Associates personnel. An aircraft was mounted in the central part of the lawn in Area 97 as an exhibit.

The first of the series of tank leaks at Area 97 was discovered in 1975, when three of the five tanks were discovered leaking. They were drained, cleaned, and filled with water. In 1978, a fourth tank was found to be leaking. This tank and the remaining fifth tank, which was the only metal

tank, were drained and filled with water, although not cleaned prior to filling with water. One to two inches of AVGAS remained on the water surface. All five tanks were removed as of 1987; the four concrete tanks were destroyed and buried in place.

Gasoline and lead have been detected in the groundwater within a 30- to 40-acre area surrounding Area 97 (Wahler/WESTDIV, 1985). An estimated 365,000 gallons of AVGAS (based on tank inventories) may have leaked into the soil, shallow groundwater, and underground utility ducts during the tank operations from the 1960s until 1978 (E & E/WESTDIV, 1983). A large amount of the fuel is thought to have evaporated.

There are reported incidents of explosion and fire in sewer and electrical manholes in the area. In 1977, an explosion occurred injuring an electrical contractor. The highest detected concentrations of combustible gas in air were found near Building 108 and west of Building 430 (Kennedy/WESTDIV, 1980).

Wastes currently are not generated at the former location of the five AVGAS tanks in Area 97, although the gasoline vapor problem has persisted.

2.1.5 Oil Refinery

An oil refinery was operated from 1879 to 1903 by Pacific Coast Oil Company. This area was located in the southeastern corner of the base, which has since been developed and occupied by other buildings.

Unknown amounts of refinery wastes (e.g., stillbottoms) and asphalt-type residues were disposed of in a 1200 × 1200 feet area during the 24 years of operation. The area was paved in the 1940s by the Navy, but vapor pressure buildup cracked the pavement. It was repaved (date unspecified) after the removal of 30 square feet of residues, the pouring of a concrete slab over the surface, and backfilling with clean fill (E & E/WESTDIV, 1983; Canonie/WESTDIV, 1988a).

"Black oil" has been encountered in drilling operations in the former disposal area of the Oil Refinery and may have locally migrated into the groundwater and shallow soils (E & E/WESTDIV, 1983).

2.1.6 Fire Training Area

This area is located on the northern perimeter of the base in close proximity to the Oakland Estuary. From the 1950s to the present, this site has served as a fire fighting training ground, a fire extinguisher discharge point, and a contraband drug burning area. An open steel tank which rests on a concrete slab (20 feet by 30 feet) is used for burning wastes. The area is partially enclosed by an earthen berm. Monthly burnings of approximately 200 gallons of fuels from plane defueling operations and waste oil have been conducted (Alliance/WESTDIV, 1987; Canonie/WESTDIV, 1988a).

Wastes in the Fire Training area include aqueous Ansulite fire-fighting foam, potassium chloride, and Purple K (fire-fighting foam). Waste fuel and oil may contain metals and PCBs. Approximately sixty to seventy (60 to 70) 30-lb potassium chloride extinguishers were discharged three to four times a year for at least 10 years. Surface soil stains have been reported (E & E/WESTDIV, 1983).

2.1.7 Building 114

Building 114 is located approximately one third of a mile south of the main gate. The western part of the building is used for administrative offices. The eastern part of the building currently is occupied by the Public Works Center that includes a paint shop and a number of maintenance areas.

In the past, Building 114 housed the majority of Public Works' shop areas. Activities and uses included woodworking, painting, paint stripping, and steam cleaning. A separator pit, located in the western corner of the courtyard of Building 114, was intended to separate sludges and floating scums

from the wastewater stream. This separator system is known to have operated inadequately (E & E/WESTDIV, 1983). Periodically, the separator pit was pumped out and the contents disposed of at the West Beach Landfill (Canonie/WESTDIV, 1988a).

Building 114 also served as the pesticide and herbicide storage and operations area for the base. Equipment and chemicals were stored at Building 114 prior to 1974. Pesticides used in the past include: chlordane, lindane, and DDT (insecticides); diuron (Telvar), monuron (Chlorvar and Telvar), bromocil, and 2,4-D (herbicides). Pesticides in current use include: glyphosphate (Roundup), simazine (Princep), diuron and bromocil (Krovar I); malathion and diazinon (insecticides); and warfarin (rodenticide) (E & E/WESTDIV, 1983).

Approximately 250 gallons per day of wastewater from steam cleaning, paint stripping, and paint spray booth operations were discharged to the storm drain and sump over an undefined period of time. Paints, solvents, and pesticides rinsed from equipment were reportedly discharged into the storm drain. The sump and storm drains emptied into the Seaplane Lagoon. No major spills have been reported (Canonie/WESTDIV, 1988a).

2.1.8 Building 5 (Plating, Paint Stripping, Cleaning, and Paint Shops)

Building 5 (B-5) is a very large (18.5-acre) building that was constructed in 1942. The variety of industrial activities conducted in Building 5 include: machining, conversion coating, painting, paint stripping, plating, reworking and manufacturing of metal parts, and maintenance and repair. Major industrial waste generators in Building 5 are plating, painting, paint stripping and conversion coating, and cleaning operations.

Plating shop activities include degreasing, caustic and acid etching, metal stripping and cleaning, and chrome, nickel, silver, cadmium, and copper plating. Wastes include rinse tank wastewater, concentrated plating bath

dumps, plating tank sludges, caustic cleaners, and cyanide stripper bath dumps (ERM-West/Aqua Resources/WESTDIV, 1987).

Since 1975, chromium process wastewaters have been discharged at an average rate of 19,200 gallons/day to the Building 5 Industrial Waste Treatment Plant (IWTP) prior to entering the main industrial sewer system. Cyanide process wastewaters were also discharged directly to the industrial sewer system at a rate of 14,400 gallons per day; however, cyanide process waters are currently treated for cyanide oxidation at the Building 5 Plating shop and heavy metals removed at the Building IWTP. Plating bath liquids and sludges are pumped out and drummed and disposed off base by private contractors; however, prior to 1970, baths were directly discharged to the industrial waste collection system or to the West Beach Landfill (E & E/WESTDIV, 1983).

Conversion coating is an intermediate step between paint stripping and painting. The conversion coat is a surface primer containing chromate, activators, and some dissolved aluminum and iron. The wastewater has a high pH and contains aluminum, chromium, and iron. Wastewater is treated at the Building 5 IWTP.

In the past, paint stripping processes utilized phenolic stripping compounds that entered the rinsewaters in large quantities. In addition to phenol, wastewaters also contained methylene chloride, chromium, oil and grease. There is also a potential for PCB contamination due to the previous use of clean-up rags containing PCBs in stripping shops (ERM-West/Aqua Resources/WESTDIV, 1987). Presently, painting stripping is no longer conducted in Building 5 with the exception of hot tank stripping in the B-5 cleaning shop. The hot tank stripper contains no phenolic compounds. The rinsewater from the hot-tank stripping operation is discharged into the Building 5 IWTP.

Cleaning shop activities included cleaning and paint stripping of parts in spray booths and dip tanks, as well as, sand-, bead-, or hull-blasting of

aircraft parts. Trichloroethane is used to degrease parts. From 1940 to the late 1970s, solvents such as carbon tetrachloride and 1,1,1-trichloroethane were used. Currently, cleaning solvents are recycled and reused. Rinsewater and paint stripping wastewaters discharge through a floor drain that connects to the industrial wastewater collection system, and contain elevated levels of phenol, chromium, suspended solids, and oil and grease. Daily wastewater discharges are 25,000 gallons.

Painting activities are confined to two bays (east and west) used for spray painting aircraft and spray booths for small parts. The wastewater contains chromium, zinc, and iron. According to NAS Alameda, this wastewater is discharged to the Building 5 IWTP for treatment only if laboratory analysis documents Total Toxic Organics to be below 2.13 ppm. If the concentration exceeds the threshold, the wastewater is disposed as hazardous waste. The average volume of wastewater generated is approximately 5,000 gallons per day.

Other miscellaneous wastes generated by operations in Building 5 and disposed of off site in 55-gallon drums are beryllium wastes, mercury contaminated rags or equipment, asbestos from aircraft insulation, various contaminated petroleum, products, and spent abrasives.

Prior to the late 1960s, wastes generated in Building 5 were disposed of at the West Beach Landfill and the 1943-1956 Disposal Area. According to DHS, from 1942 to 1975, at least 18,000 tons of waste were generated at Building 5. Tanks regularly overflowed and entered drains which discharged untreated to the industrial waste collection system. There have been possible releases of a wide variety of organic and inorganic chemicals to the soil beneath the building or to soil and groundwater from industrial sewers (E & E/WESTDIV, 1983; Canonie/WESTDIV, 1988a; ERM-West/Aqua Resources/WESTDIV, 1987).

2.1.9 Building 360 (Plating, Engine Cleaning, Paint, and Paint Stripping (Shops)

Building 360 is located near the eastern perimeter of the base, occupying approximately 5-½ acres of land. In operation since 1954, Building 360 houses specialized shops for the repair and testing of aircraft engines. These shops conduct rework milling, maintenance, repair and assembly, and testing of both jet turbine and propeller aircraft engines.

The engine cleaning shop has been used from 1954 until the present. In early 1979, chemicals seeped through the shop floors and contaminated soil in the crawl space. The contaminated area measures approximately 135 feet by 155 feet, and was contaminated to an unknown depth. Chemicals which leaked included caustics, alkaline permanganate, cleaning solvent, hydrochloric acid, nitric acid, paint remover, phosphoric acid, rust corrosion remover, and sodium hydroxide. In June 1982, the top 4 inches of contaminated soil were removed and a layer of plastic was installed under the shop to protect maintenance personnel who work in the crawl space. Some chemical leakage has occurred since the installation of the plastic (Alliance/WESTDIV, 1987).

Plating shop operations include paint stripping by blasting; chrome, lead, silver, and nickel stripping; etching and anodizing, and chrome, nickel, lead, tin, silver, and copper plating. Cyanide process wastewater typically contains cyanide (4 ppm), nickel (6 ppm), total solids (210 ppm) and COD (330 ppm) at pH levels near 8. The discharge rate is approximately 5,400 gallons per day. Chromium wastewaters are discharged to the Building 360 IWTP at a rate of 2,700 gallons per day. Chromium wastewater typically contains total chromium (40 ppm). Prior to 1975, wastewater from the plating operations was discharged directly to the Seaplane Lagoon (E & E/WESTDIV, 1983). Soil under the Building 360 plating shop has high alkalinity and a high cyanide content.

The paint shop maintains four paint booths for painting small amounts of machine parts. Degreasers in the paint shop primarily use 1,1,1-trichloroethane; however, prior to 1970, carbon tetrachloride was also

used. Currently, TCA is recycled until exhausted, at which time it is drummed with paints, paint sludges, and paint containers and disposed of off base.

In the past, the cleaning and blasting shop cleaned metal parts using baths of phenolic-based cleaners, alkaline-type cleaners, rust remover, descaling compounds, and caustics. Phenolic cleaners are not currently used. Wastes include spent process baths and solvents, waste dye penetrants, and machine coolants. Process baths at one time were discharged into the sewer system and solvents were disposed of at the West Beach Landfill. Since the 1970s, solvents have been recovered and recycled. Waste materials are moved to temporary or permitted storage facilities and disposed of off base. All wastewater from the Building 360 clean and blast shop is presently discharged into the industrial sewer after neutralization by pH adjustment and conformity with East Bay Municipal Utility District pretreatment requirements.

Activities in the engine rework shop generate various contaminated petroleum products.

2.1.10 Building 410

Building 410 is located approximately a third of a mile west of the south gate. All wastes generated in Building 410 are from the paint stripping activities. Wipe-down solvents such as ethyl acetate are used to remove rubber and other special coatings. Soiled rags, empty containers, wastewater strainings, and other miscellaneous solid hazardous wastes are drummed and properly disposed of off site.

Paint strippers contain large amounts of phenol, methylene chloride, chromium, and detergents. There may have been spills and sewer leaks containing oils, paints, paint strippers, and detergents at this site.

The wastewater at this site contains oil, paint, paint skins, detergents, and stripper. It is strained to remove paint skins and discharged at a rate of approximately 16,000 gallons per day to the Building 410 IWTP.

Prior to the construction of Building 410's IWTF in 1973, wastewater was discharged to the collection system without treatment. Wastewaters from this building area are known to contain high concentrations of chromium, phenols, and methylene chloride (ERM-West/Aqua Resources/WESTDIV, 1987).

2.1.11 Building 400 and 530 (Missile Rework Operations)

The missile rework operations currently are housed in Building 530. Prior to 1972, Building 400 housed the missile rework operations. Operations are described as small scale and include complete rework of missiles, their guidance systems and their charges, including disassembly, parts cleaning, metal grinding, welding, fabricating, paint stripping, and painting (ERM-West/Aqua Resources/WESTDIV, 1987).

Building 400: Building 400 is located at the northwestern corner of the Seaplane Lagoon. A small paint stripping, fiberglassing, and aircraft parts cleaning operations is currently in this area.

Prior to 1972, wastes generated from missile rework operations included: paint sludges; metal shavings; paint strippers; cleaning solvents (1,1,1-trichloroethane and carbon tetrachloride), methyl ethyl ketone; waste resin and catalysts from building poly/fiber structures; testing fluids; and miscellaneous waste oils and greases. These wastes were disposed of at the West Beach Landfill. Wastewater was discharged directly into the industrial waste collection system since no pretreatment was occurring at that time. No spills are documented (ERM-West/Aqua Resources/WESTDIV, 1987).

Building 530: Building 530 is located west of the south gate and the current site for missile rework operations. Current waste handling procedures are tightly controlled, with all wastes and paint stripping baths disposed of off-site. Wastes generated at Building 530 are the same as noted for Building 400 above (Canonie/WESTDIV, 1988a).

2.1.12 Building 14 (Test Shop)

Building 14 is located adjacent to the eastern side of the Seaplane Lagoon. The building currently houses two active engine testing chambers; the remainder are inactive. The laboratories which occupy the second floor of the building have reported small mercury spills from manometers and thermometers. Canonie/WESTDIV (1988a) conducted a site visit in March 1988 and noted no signs of visible contamination, but identified a 4-foot square area on the floor which will be further investigated for mercury contamination.

Mercury wastes were originally disposed of at the West Beach Landfill or the 1943-1956 Disposal Area, and some spills may have entered the industrial waste collection system or the storm sewer system (Alliance/WESTDIV, 1987).

2.1.13 Buildings 301 and 389

Building 301 and the foundation of Building 389 are located approximately 500 feet inland from the Estuary (Oakland Inner Harbor). This area was used to store discarded electrical equipment such as transformers and other machinery. Prior to 1974, electrical transformers were stored on bare ground primarily north and west of Building 389. During a site visit in March 1988, Canonie/WESTDIV (1988a) noted a small area of stained bare ground immediately north of Building 301. Several 55-gallon drums of hydraulic fluid are currently stored in Building 301.

Leakage of PCB oil has been reported. Some PCB oil was routinely drained from transformers and spread on the ground to control weed growth. Approximately 200 to 400 gallons of PCB oil may have been stored at the site at any one time (ERM-West/Aqua Resources/WESTDIV, 1987).

2.1.14 Cans C-2 Area

The Cans C-2 Area is located in the southeastern corner of the NAS Alameda. Since approximately 1963, this 6.5 acre area has been used as a

storage area. The main storage yard is 3 acres along the western side of the site and is the area of focus in this study area. Materials stored in this fenced area include disused plating and paint stripping tanks; electrical equipment; aircraft parts; and miscellaneous waste materials. The main storage yard currently is unpaved, although much of it is covered with perforated-steel temporary runway-plates (Canonie/WESTDIV, 1988a).

Waste materials stored in the Cans C-2 storage yard contain solvents, paints, paint strippers, organic chemicals, PCBs, acids, and bases. Weed control by spraying PCBs was practiced until 1963. A PCB transformer reportedly leaked and 10 cubic yards of PCB-contaminated soil were removed in 1982 (ERM-West/Aqua Resources/WESTDIV, 1987).

2.1.15 Station Sewer System

The Station Sewer System consists of the industrial and storm sewers serving Buildings 5, 360, 410, 400, 14, and 10. From 1943 to 1972, untreated wastewaters flowed directly into the industrial sewer which emptied into either the Seaplane Lagoon or the Estuary (Oakland Inner Harbor). Since 1956, the station sanitary sewer system has been discharging to the EBMUD. Between 1972 and 1975 untreated industrial waste water was discharged to the EBMUD system. Since 1975 industrial wastewater from Building 5, Building 360, and Building 410 have been pretreated prior to discharge to the sanitary sewer system. Storm drains flow directly into either the Seaplane Lagoon or the estuary (Canonie/WESTDIV, 1988a).

There currently are three industrial waste treatment plants (IWTP) at NAS Alameda, which are located in Buildings 5, 360, and 410. All of the industrial waste treatment plants are designed to provide chromium reduction, neutralization, metal precipitation, and solids removal. Following treatment, the industrial waste water is discharged to the sanitary sewer system which empties into the East Bay Municipal Utilities District's Treatment Plant. The chemical sludges generated by these industrial waste treatment plants are

disposed of off site as hazardous wastes. An estimated 17 million gallons of wastewater are treated annually (ERM-West/Aqua Resources/WESTDIV, 1987).

The Building 5 IWTP treats chromium containing wastewaters from the Building 5 plating and conversion coating processes and rinsewater from the parts cleaning shop. Cyanide containing wastewaters are no longer released to the sanitary sewer without treatment.

The Building 360 IWTP treats only chromium wastewaters from the Building 360 plating shop. Cyanide paint stripping and conversion coating wastewaters are discharged to the sanitary sewer without treatment (E & E/WESTDIV, 1983).

The Building 410 IWTP treats paint stripping wastes. Paint skins are removed by screening and are disposed of off site.

Unknown amounts of wastewater from plating bath dumps, paints, paint strippers, pesticides and herbicides, waste fuels and oils, solvents, and possibly PCB-contaminated oils were discharged into the sewer and storm drain systems from 1943 to the present (E & E/WESTDIV, 1983).

2.1.16 Seaplane Lagoon

This body of water has a surface area of 110 acres, is 12 to 20 feet in depth and is almost entirely enclosed by seawalls. The southwestern end of the lagoon opens to San Francisco Bay. From 1943 to 1975, the lagoon served as a receiving basin for an estimated 300 million gallons of wastewaters from five industrial and storm sewer outfalls.

Wastewaters released to the Seaplane Lagoon prior to 1975 were contaminated with metals, solvents, paints, detergents, acids, alkalies, mercury, oil, grease, pesticides, PCBs, and fuel. Ships docked at the piers south of the entrance to the Seaplane Lagoon discharged wastewater which could have been swept into the lagoon by tidal action. During the 1960s and 1970s,

paint from the bottom small boats anchored in the lagoon was reported to dissolve (Alliance/WESTDIV, 1987).

In 1981, 21,000 cubic yards of sediments were dredged from the southeast side of the lagoon and disposed of at the West Beach Landfill. Other dredging activities have been of small quantities (E & E/WESTDIV, 1983).

2.1.17 Estuary (Oakland Inner Harbor)

The estuary is a 2.2 mile harbor channel bordering the northern edge of NAS Alameda. From 1943 to 1978, the estuary received approximately 150 million gallons of untreated industrial and nonindustrial wastewater through the storm water sewers. The wastewater contained organics, metals, detergents, oils, and pesticides (Canonie/WESTDIV, 1988a).

2.1.18 1943-1956 Disposal Area

The 1943-1956 Disposal Area is located in the northwestern corner of the station and occupies an area of 120 acres. The estuary (Oakland Inner Harbor) lies along its northern boundary and the San Francisco Bay along its western boundary. The majority of the area is now paved or covered with soil and includes runways, taxiways, a picnic ground and baseball diamond, jogging track, two ammunition storage facilities, and a pistol range (E & E/WESTDIV, 1983). During a site visit by Clement personnel in September 1988, the picnic ground and baseball diamond located in the former 1943-1956 Disposal Area appeared to be permanently closed.

The 1943-1956 Disposal Area was a landfill from 1943 to 1956 and received an estimated 15,000 to 200,000 tons of waste. The disposal method reportedly used consisted of digging trenches to the water table, filling them with wastes, and compacting the material with a bulldozer. Cover material was applied on an irregular basis (Canonie/WESTDIV, 1988b).

Wastes disposed include low level radiological waste, waste oil, paint wastes, solvents, cleaning compounds, as well as oil aircraft engines, garbage, scrap metal, and construction debris (Alliance/WESTDIV, 1987).

2.1.19 West Beach Landfill

The West Beach Landfill is located in the southwestern corner of the NAS Alameda and encompasses about 110 acres. The San Francisco Bay is located along its western and southern boundary. Current site conditions in the landfill are based on a site visit by Clement personnel in September 1988. The site is currently vegetated with an approximately 5-acre wetland. A 5-foot high earthen berm surrounds the landfill. The landfill area is enclosed by a locked fence.

The West Beach Landfill served as a disposal site for NAS Alameda from approximately 1952 through March 1978. In addition, other naval installations which included Oak Knoll Naval Hospital, Naval Supply Center, Oakland, and Treasure Island disposed wastes in the West Beach Landfill. The disposal method reportedly used consisted of digging trenches to the water table, filling them with wastes, and compacting the material with a bulldozer. Cover material was applied on an irregular basis (Canonie/WESTDIV, 1988b).

Wastes reportedly disposed of in the West Beach Landfill include solvents, PCBs, plating wastes, metals, pesticides, ordnance, low level radioactive waste, infectious waste, acids, oily waste and sludges, paints, strippers, thinners, mercury, tear gas agents, batteries, asbestos, and creosote. Approximately 992,800 tons of municipal wastes including 30,000 to 300,000 tons of potentially hazardous wastes were estimated to have been disposed in the West Beach Landfill between 1958 and 1978.

2.1.20 Yard D-13

Yard D-13 is located southwest of Building 360 and 1500 feet east of the Seaplane Lagoon. It is a 1.5-acre drum storage yard that is paved and enclosed by a fence. The area was most recently repaved in 1988.

Potentially hazardous wastes generated by base activities are stored in 55-gallon drums in Yard D-13. Wastes stored included alkalies, Poison B materials such as beryllium wastes, endosulfan, and endrin, acids, acid oxidizers, and flammable and combustibles grouped by chemical class in rows separated by berms (Canonie/WESTDIV, 1988a).

2.2 ENVIRONMENTAL CHARACTERISTICS

The potential impacts of contaminants at any site partially depend on the ecological characteristics of the site and the surrounding areas. Contaminant migration off site is influenced by local meteorology, topography and surface drainage, hydrology, and geology as well as other site characteristics. These are summarized briefly below for NAS Alameda.

2.2.1 Meteorology

Temperatures in the Alameda area are generally moderate. Freezing temperatures rarely occur. Rainfall averages approximately 20 inches per year with the majority of the precipitation occurring from October to May. Winds in the San Francisco Bay area generally blow from the west to the east and are rarely of gale force or greater. Heavy fogs occur on an average of 21 days per year. These fogs impair visibility for navigation in the surrounding San Francisco Bay and Oakland Inner Harbor Channel an average of less than 100 hours per year (E & E/WESTDIV, 1983).

2.2.2 Topography and Surface Drainage

The island of Alameda in Alameda County is located along the eastern shore of San Francisco Bay just east of the City of Oakland. The City of San Francisco is located west of Alameda on the opposite side of the Bay (See Figure 2-1). The island lies on a flat topographic profile with elevations ranging from zero to 30 feet above mean sea level. The average land elevation of the island is approximately 20 feet. NAS Alameda occupies 2,570 acres on the western tip of the island, where the topography is slightly flatter. Land elevation on the station ranges from 10 to 15 feet above mean sea level. Precipitation and other runoff is either evapotranspired back to the atmosphere, discharged to a sewer system, filtered down to the water table, or drained into the Bay (E & E/WESTDIV, 1983).

2.2.3 Soils and Geology

In the early 1900s, the island of Alameda extended as far west as what is now the eastern border of NAS Alameda. During this time, most of the area currently occupied by NAS Alameda was marshlands characterized by numerous drainage channels and sloughs. The marshlands were formed as the very fine particles suspended in San Francisco Bay waters settled onto the shallow Bay floors. These marine deposits are known as the Bay Mud Formation (E & E/WESTDIV, 1983).

In the 1920s, the marshlands were filled with six to eight feet of material dredged from the Bay and the Oakland Inner Harbor Channel. The fill material was moderately to poorly compacted and is characterized as a silty sand with low to moderate compressibility. These coarse soils have a low water holding capacity, allowing water to easily migrate to and settle on the clayey Bay Mud. Over the majority of the station, these soils provide adequate drainage. However, near the San Francisco Bay shoreline, only 20 to 60 inches of fill exists. As a result, these areas have poor drainage capabilities (E & E/WESTDIV, 1983).

Beneath the fill material, Bay Mud extends to depths of 25 to 120 feet below ground surface. The Bay Mud Formation consists of dark gray to olive gray organic clay that is frequently water saturated. The clay is highly plastic and compressible and often has a strong odor. The deposit is generally interlayed with silt and sand lenses (Canonie/WESTDIV, 1988b).

Substantial amounts of alluvial and aeolian soils were deposited prior to the Bay Mud formation during a period when the sea level was at a much lower elevation. These deposits consist of silty clays to clayey sands and are considered part of the Merrit Sand Formation (Canonie/WESTDIV, 1988b).

Beneath the Merrit Sand Formation are three formations known as: (1) the Posey; (2) the San Antonio; and (3) the Alameda Formations. These formations were deposited during interglacial periods and consist of clayey sand to sandy clay. The uppermost formation of the three is the Posey Formation. This layer is a sandy clay with moderately low permeability. Beneath the Posey is the San Antonio Formation. This layer is a moderately stiff silty clay which serves as a competent aquitard between the Merrit Sand Formation and the underlying Alameda Formation. The Alameda Formation is the deepest of the three formations and is considered an aquifer. The layer consists of green to gray sand, sandy clay and clay with some fine gravel (Canonie/WESTDIV, 1988b).

The bedrock lying beneath the site is comprised of an assemblage of volcanics, meta-sandstones, and a melange of sandstone, shale, chert, and serpentinite (Canonie/WESTDIV, 1988b). An off-site exploration boring located approximately one mile northwest of NAS Alameda encountered bedrock at an elevation of 433 feet below mean sea water level. The encountered bedrock was a yellow shale (E & E/WESTDIV, 1983).

No earthquake faults traverse NAS Alameda. The nearest fault is the Hayward Fault, located six miles east of the base. The San Andreas Fault is located 12.5 miles west of the base (E & E/WESTDIV, 1983).

2.2.4 Surface Water

The island of Alameda is located on the eastern portion of San Francisco Bay. The Bay is used for navigation, water contact recreation, and fishing. Seaplane Lagoon is located on the southern portion of Alameda NAS and has a pier for recreational fishing. The Oakland Inner Harbor Channel is located along the northern shoreline of the station. The Channel is used for recreational and navigational purposes. The Oakland Estuary is located in this Channel. There are no natural surface water streams or ponds located on NAS Alameda.

2.2.5 Hydrogeology

The water table beneath NAS Alameda is first encountered at elevations of 4 to 8 feet above the lower low tidal elevation, corresponding to depths ranging from approximately 2 to 11 feet below ground surface. The groundwater flows toward San Francisco Bay and the Oakland Inner Harbor Channel at a rate of approximately 15 gallons per day per foot of shoreline. The annual rainfall is 20 inches, with approximately four inches of the rainfall contributing to the water table. Because of the high permeability of the soils and the relatively steep hydraulic gradient, tidal influences do not significantly disturb the water table beyond 25 feet from the shoreline (E & E/WESTDIV, 1983).

There are two aquifer units located beneath the shallow water table: the Merrit Sand and the Alameda Formation. The shallow water table and the Merrit Sand are separated by the Bay Mud Formation, which is approximately 25 feet thick. Along the western edge of NAS Alameda, the layer of Bay Mud may be as thick as 70 feet. The Merrit Sand and Alameda Formations are separated by an aquitard which is approximately 250 feet thick. The aquitard consists of sandy silty clay and stiff silty clay of low permeability (Canonie/WESTDIV, 1988b).

Two wells are known to have been in operation at NAS Alameda. One well, the Pan American Well, is a 500-foot deep well located approximately 1,500 feet east of the 1943-1956 Disposal Area. The other well, the Army Well, is 353 feet deep, and is located east approximately 8,000 feet east of the West Beach Landfill. Canonie/WESTDIV, (1988b) reported that the Pan American Well currently is out of service, and the Army Well currently is used for landscape irrigation. The Pan American well was abandoned with the pump and associated plumbing intact.

The Pan American well was constructed prior to August 25, 1941, and was reportedly used to a depth of 447 feet. The well has been out of service since 1968 except for a pump test conducted in 1977. Groundwater was withdrawn between 275-280 feet, 320-345 feet, 385-387 feet, and 439-444 feet below ground surface, which corresponds to the middle and lower sections of the Alameda Formation. An approximately 150-foot layer of clay in the upper Alameda Formation isolated underlying fresh water aquifers from overlying brackish aquifers. Chemical analysis of a groundwater sample from the Pan American well in 1977 indicated 0.011 mg/l of mercury which exceeded the 0.002 mg/l standard of both the EPA Maximum Contaminant Level and the California applied action level. The concentration of the remaining chemicals did not exceed current primary drinking water standards. The manganese concentration of 0.07 mg/l exceeded the current 0.05 mg/l standard of the California secondary drinking water MCL. The total dissolved solids (TDS) concentration of 588.62 mg/l exceeded the current 500 mg/l recommended California secondary drinking water MCL, but had not exceeded the recommended upper limit of 1000 mg/l. The concentration of the remaining chemicals did not exceed current secondary drinking water standards. No historical chemical analyses of the groundwater from the Army Well were recorded. The present concentration of mercury in the Pan American and Army Wells are not known, but will be addressed by Canonie in future field work. The complete inorganic analysis is shown in Appendix A (Hydro-Search/Navy Public Works, 1977).

An additional well reported by Alameda County's well inventory at NAS Alameda is located east of the Army well and approximately 9,500 feet east of

the West Beach Landfill. This well was 376 feet deep and was abandoned in place. Past uses of the well was not documented. (Canonie/WESTDIV, 1988b). The integrity of the construction of this well, the Pan American well, and the Army well will be investigated by Canonie in future field work. This will indicate if these wells were potential vertical conduits for the migration of chemicals from shallow aquifers or the surface. Chemical analyses of groundwater from these wells will confirm if elevated levels of mercury exist in the deep aquifer beneath NAS Alameda.

The Alameda County Flood Control District office supplied the following information on current groundwater use in the area of NAS Alameda. The majority of the wells throughout the City of Alameda obtain water from the Merrit Sand and Alameda Formations. The water is used for irrigational and industrial purposes and is not used as a municipal water supply. The City of Alameda obtains drinking water from the East Bay Municipal Utility District which draws water from the Mokelumne Watershed, located more than 100 miles east of NAS Alameda in the Sierra Nevada Mountain Range. A well at Alameda High School and another at a public golf course is used for irrigation. Industrial water uses of the local groundwater include car wash and laundromat services.

2.3 IDENTIFICATION OF SITE-RELATED CHEMICALS

Chemicals detected in environmental media sampled during past sampling efforts (groundwater, soil, surface water, sediment, and air) have been identified and their distribution evaluated. Chemicals related to past activities at the site which were not analyzed for will be proposed to be evaluated in the final PHEE.

Available data are summarized by presenting frequencies of detection, geometric means and maximum values for each chemical detected in an individual medium by study area. Geometric means rather than arithmetic means were calculated since most environmental contaminants are log-normally distributed (Dean, 1981; Ott, 1988). Because of the absence of data for some study areas and the limited volume of data in the other study areas, the tables presented in this preliminary PHEE must be considered preliminary, and additional data from the RI will be incorporated in the final PHEE. In the present report,

only samples with measured chemical concentrations were used in calculating the geometric mean.

Currently available sample data on the extent of chemical contamination at the site is outlined below separately for each of the 20 study areas considered in this PHEE. Data for 6 of the 20 study areas were taken from the Draft Report verification step of the Confirmation Study for NAS Alameda (Wahler/WESTDIV, 1985). Data for the West Beach Landfill study area were from two studies conducted by Harding and Lawson (HLA/WESTDIV 1978, 1983). Additional data for Area 97 were taken from a Subsurface Fuel Contamination Study conducted by Kennedy Engineers (Kennedy/WESTDIV, 1980). Sediment data for the Oakland Harbor were taken from a 1988 study completed by the U.S. Corps of Engineers. Known or suspected contamination of soil, air, groundwater and surface water is discussed separately.

2.3.1 Building 41 (Maintenance Area)

Environmental sampling results are not available for this area.

2.3.2 Buildings 459, 547, and 162 (Service Stations)

Environmental sampling results are not available for this area.

2.3.3 Building 10 (Power Plant)

Environmental sampling results are not available for this area.

2.3.4 Area 97

Two rounds of sampling occurred in Area 97. Kennedy/WESTDIV (1980) installed 18 monitoring wells and sampled each for gasoline hydrocarbons. In addition, 17 subsurface soil samples were collected and analyzed for gasoline hydrocarbons by Kennedy/WESTDIV. Wahler/WESTDIV (1985) installed three additional monitoring wells in 1983 and collected groundwater samples from the

three new wells and eleven of the pre-existing monitoring wells. In addition, residual water samples were collected from four of the five abandoned fuel tanks and one water sample was collected from groundwater that seeped into a trench excavated along the western border of Area 97. Water samples collected in the second round of sampling were analyzed for gasoline hydrocarbons and lead. Frequencies of detection, geometric means, maximum values, and sample location of maximum values of each chemical detected by medium are summarized in Table 2-1. Sample locations are shown in Figure 2-3.

The source of contaminants appeared to be five 100,000-gallon aviation gasoline (AVGAS) tanks removed in 1987 (Kennedy/WESTDIV, 1980). These tanks are known to have leaked prior to removal. The following media-specific data are available:

Soil: An initial study of Area 97 (Kennedy/WESTDIV, 1980) reported elevated concentrations of AVGAS in subsurface soils at locations OW-1, OW-16, and OW-23 at concentrations of 1100 mg/kg, 9200 mg/kg, and 7600 mg/kg, respectively. The remaining 14 soil samples contained less than the analytical detection limit (720 mg/kg) of gasoline hydrocarbons. The discrete soil samples generally ranged in depth from two to seven feet below ground level. No surface soil samples were collected. The source of contaminants appeared to be Tanks A and B in the northwest corner of Area 97.

Air: Electrical duct manholes, sanitary sewer manholes, and storm drain manholes in the proximity of Area 97 were tested for the presence of fuel vapors by Kennedy/WESTDIV (1980). A portable fuel vapor "sniffer" with a vapor intake tube inserted through a vent hole in the manhole cover measured the vapor concentrations in parts per million (volume) hydrocarbons as hexane. Elevated levels of fuel vapor were detected in manholes along Atlantic Avenue (up to 1,500 ppm) immediately west of Area 97, immediately northwest of Tank A (1,200 ppm), and in a sanitary sewer adjacent to the Seaplane Lagoon along Fifth Avenue (100 ppm to greater than 10,000 ppm). The source of the fuel vapors detected near the Seaplane Lagoon was not identified (Kennedy/WESTDIV, 1980).

Table 2-1
Area 97 Sampling Results (1979 and 1983)

Chemical	Soil Borings (2 to 7 ft Deep) Freq. of Detection	Geom Mean (mg/kg)	Maximum Conc. (mg/kg)	Location of Maximum Conc.	Groundwater Freq. of Detection	Geom Mean (mg/l)	Maximum Conc. (mg/l)	Well Location of Maximum Conc.	Open Trench Water Sample (mg/l)	AVGAS Tanks # detects/ # samples	Geom Mean (mg/l)	Maximum Conc. (mg/l)	Location of Maximum Conc.
Gasoline Hydrocarbons	3/17	4300	9200	OW-16	8/31	7.6	41	OW-23	3900	1/4	-	2400	E
Lead	na	na	na	na	12/14	1.2	210	OW-6	2.5	4/4	0.22	3.1	E
Oil & Grease	na	na	na	na	1/1		1410	OW-6	na	na	na	na	na

Data taken from Wahler/WESTDIV (1985) and Kennedy/WESTDIV (1980).

Geometric means of detected samples only.

Detection limits were as follows: Gasoline hydrocarbons, 720 mg/kg (soil), and .02 to 3 mg/l (water); and lead, .01 mg/l (water).

na = not analyzed

Visible free product noted in 1983 sampling of open trench and wells OW-3, OW-6, and OW-14.

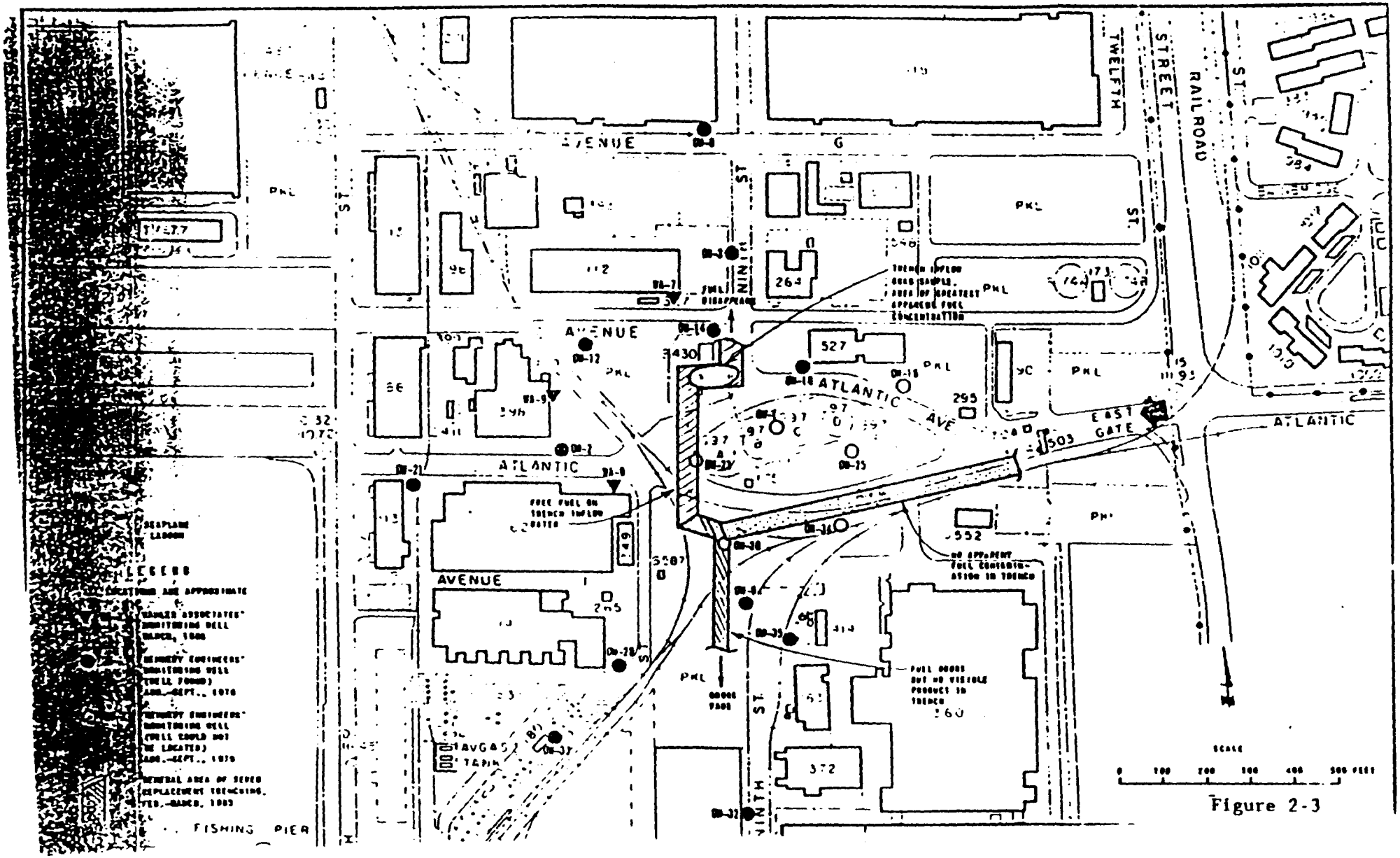

AVENUE G
TWELFTH STREET
RAILROAD
ATLANTIC AVE
ATLANTIC
AVENUE
NINTH ST
EAST GATE
PKL
AVGAS TANK
FISHING PIER
SEAPLANE LAGOON
SCALE
0 100 200 300 400 500 FEET

Figure 2-3

Groundwater: Monitoring wells were generally screened from about 5 to 17 feet below ground level. Elevated concentrations of lead were present in twelve of the fourteen groundwater samples analyzed in 1983. It is unknown whether total or dissolved lead levels were reported. The higher concentrations were found in wells OW-6 (210 mg/L), OW-21 (19 mg/L), OW-32 (5.5 mg/L), OW-3 (5.3 mg/L), and OW-14 (2.7 mg/L). The remaining seven monitoring wells each contained less than 1 mg/L of lead. The water sample from the excavated trench contained 2.5 mg/L of lead.

Elevated levels of AVGAS were found in the monitoring wells OW-1, OW-16, OW-14, OW-23, and WA-7 located near the former location of tanks A and B and to the north and northwest of Area 97. Visible AVGAS was observed floating on top of the water during the 1983 sampling round in the sewage excavation trench west of Area 97, and in monitoring wells OW-3, OW-6, and OW-12. A sample of water collected from a trench excavated south of Building 430, in the vicinity of Building 109, contained the highest concentration (3900 mg/L) of gasoline hydrocarbons reported in the 1983 sampling round. This suggests that: (1) although the AVGAS tanks were abandoned, a local source or reservoir still existed in the immediate vicinity of Area 97 in 1983, and (2) gasoline hydrocarbons may have migrated along underground utility lines. The plume of contaminants appears to be moving northwest to the north of Area 97, with the furthest extent of the plume's migration not documented (Well OW-8 was dry in the 1983 sampling round; Wahler/WESTDIV, 1985).

Well OW-6 contained a black oily substance containing high-boiling hydrocarbons not typically found in gasoline or diesel fuel. The oil and grease concentration in Well OW-6 was 1410 mg/L in the 1979 sampling round. The potential source or sources of the contaminants, in particular the elevated lead levels (210 mg/L) in Well OW-6 was not identified by Wahler/WESTDIV (1985) or Kennedy/WESTDIV (1980).

Kennedy/WESTDIV (1980) reported that the direction of groundwater movement from Area 97 was predominantly to the west and the northwest. The underground utilities along Atlantic Avenue, south of Area 97, appear to

create a local groundwater depression. The utility lines may act as a physical barrier to AVGAS movement, or the utility lines may act as a conduit for AVGAS migration toward the Seaplane Lagoon.

Other Samples: Gasoline hydrocarbons were detected in only one of the four residual water samples (Tank E, 2400 mg/L) collected from the abandoned AVGAS tanks in 1983. Lead was present in all four residual water samples with the highest concentration in Tank E (3.1 mg/L). These analytical results confirm that leaking AVGAS tanks were one potential source of the plume of gasoline hydrocarbons and lead contaminants.

2.3.5 Oil Refinery

Environmental sampling results are not available for this area.

2.3.6 Fire Training Area

Environmental sampling results are not available for this area.

2.3.7 Building 114 (Pest Control Areas)

Environmental sampling results are not available for this area.

2.3.8 Building 5

Environmental sampling results are not available for this area.

2.3.9 Building 360

Wahler/WESTDIV (1985) reported that the Environmental Research Group collected an unspecified number of soil samples in the crawl space beneath Building 360. The parameters analyzed, analytical methods, and detection limits were not specified. The soils collected from the crawl space contained cyanide with a maximum concentration of 118 mg/kg (ppm). One of two soil

samples analyzed for trichloroethylene (TCE) and 1,1,1-trichloroethane (TCA) contained a trace of TCA, but TCE was not detected. These same two soil samples were analyzed for various acids and hydroxides, which were detected, but the concentrations were not specified. The soil samples were alkaline, with most of the pH values between 9.1 and 9.8.

2.3.10 Building 410

Environmental sampling results are not available for this area.

2.3.11 Buildings 400 and 530 (Missile rework operations)

Environmental sampling results are not available for this area.

2.3.12 Building 14 (Test Shop)

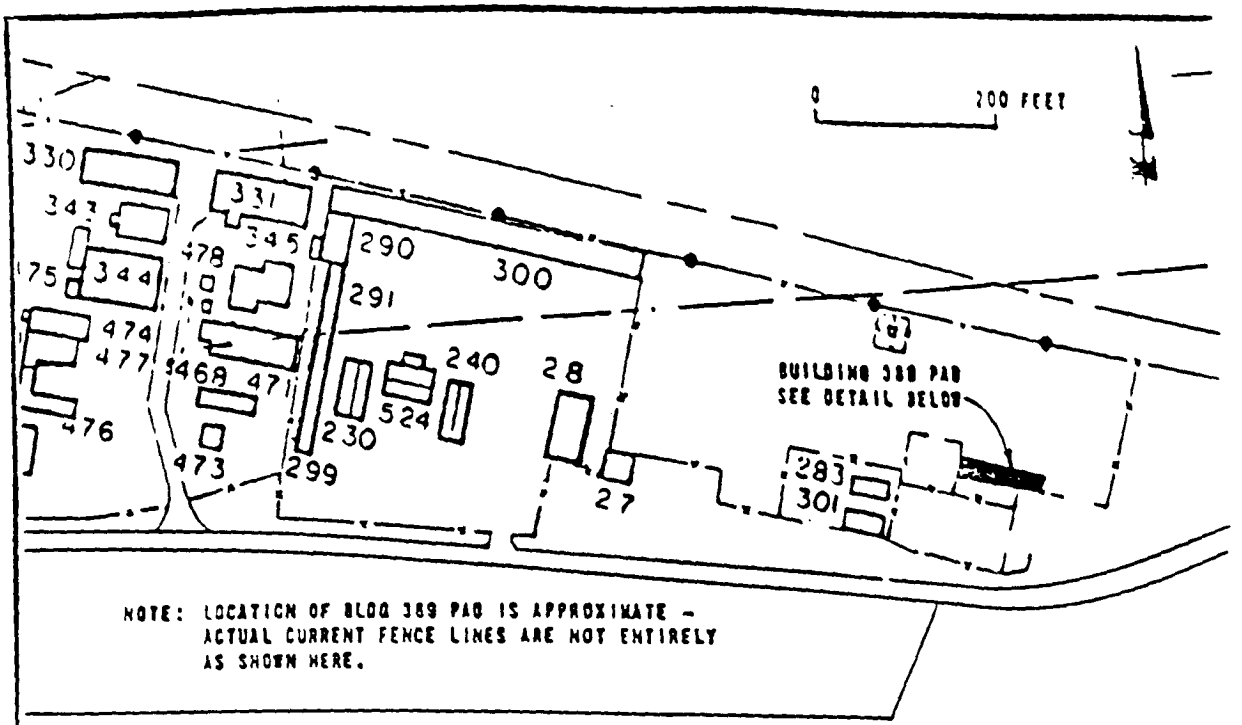
Environmental sampling results are not available for this area.

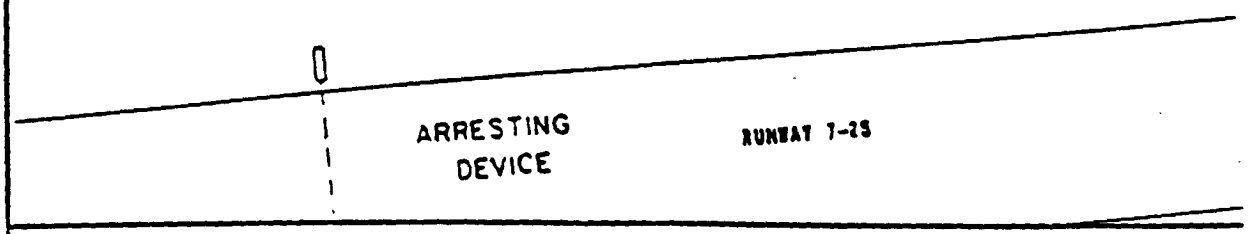
2.3.13 Buildings 301 and 389 (Transformer Storage Area)

Ten of 12 shallow soil grab samples collected beneath the asphalt or from potholes in the former transformer storage area adjacent to the former location of Building 389 were analyzed for PCBs (Wahler/WESTDIV, 1985). The locations of the soil samples are shown in Figure 2-4. PCBs were present above the detection limit (1 mg/kg) in four of the ten samples. Sample locations with detectable concentrations of PCBs (measured as Arochlor 1260) were: J-1 (3 mg/kg), J-3 (3 mg/kg), J-5 (1 mg/kg), and J-8 (3 mg/kg).

2.3.14 Cans C-2 Area

Soil: Eleven grab soil samples, ten surface (0.5 ft) soils and one subsurface (6 to 6.5 ft) soil, were collected in the Cans C-2 Area in 1984-1985 at the locations shown in Figure 2-5. Soil samples were analyzed for seventeen metals, organochlorine pesticides, PCBs, chlorophenoxy herbicides,





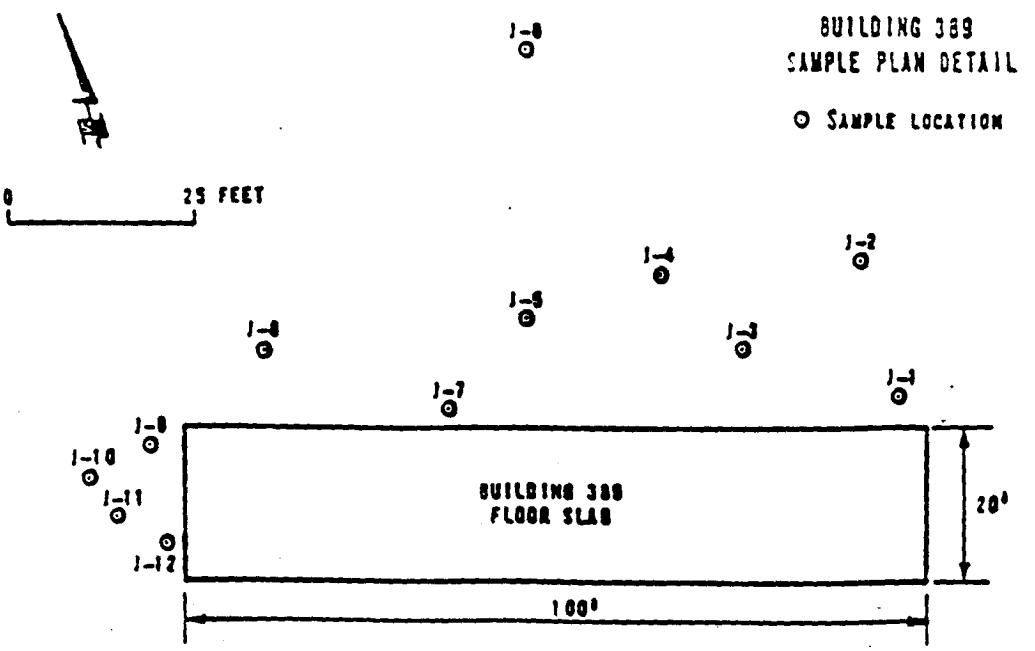
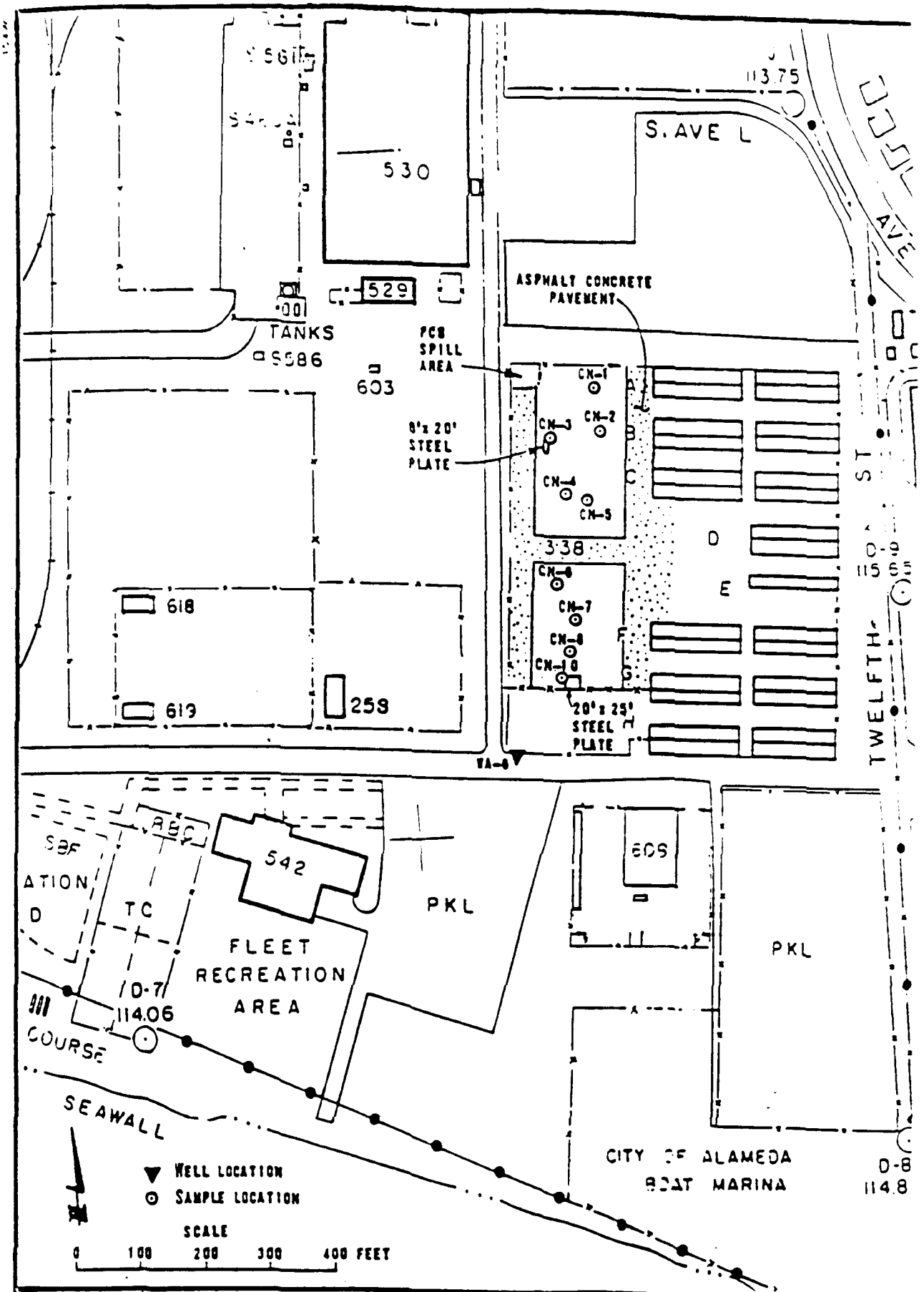


Figure 2-4 SAMPLE LOCATIONS BUILDINGS 301 & 389



Wahler | CONFIRMATION STUDY 2-35 | Figure 2-5 WELL AND SAMPLE LOCATIONS CANS AREA

and gasoline hydrocarbons. Table 2-2 presents frequencies of detection, geometric means, maximum values and sample locations of maximum values of each chemical detected, by medium (Wahler/WESTDIV, 1985). Background soil samples were not collected.

Ten of the seventeen inorganics analyzed were detected in the surface and subsurface soil samples. Inorganics detected in the soils were barium, cadmium, chromium, cobalt, copper, lead, mercury, nickel, vanadium, and zinc. There were no organic chemicals detected in the soil samples, except for a trace of gasoline hydrocarbons in sample CN-5 (0.05 mg/kg). The soil pH ranged from 5.9 to 7.8, with most values less than 7.0 (Wahler/WESTDIV, 1985).

Groundwater: One monitoring well (WA-6) was installed in the southwestern corner of the Cans C-2 Area in 1983. The well was screened from 5 to about 25 feet below the ground level. Well WA-6 is reported to be downgradient from the Cans C-2 area (Wahler/WESTDIV, 1985). One groundwater sample collected in January 1985 was analyzed for seventeen metals, organochlorine pesticides, PCBs, chlorophenoxy herbicides, and gasoline hydrocarbons. Chromium (total) and 2,4-D were the only chemicals detected in the groundwater sample, at concentrations of 130 μg/l and 2 μg/l, respectively, as shown in Table 2-2. Alliance/WESTDIV (1987) reported that groundwater samples collected in 1986 contained elevated concentrations of 1,2-dichloroethylene and endrin, but did not report concentrations.

2.3.15 Station Sewer System

Environmental sampling results are not available for this area.

2.3.16 Seaplane Lagoon

Sediment: Shallow sediment samples (estimated depth of 0 to 0.5 ft) were collected at eight locations within the Seaplane Lagoon and at two potential background locations southwest of the lagoon in the channel. Sediment samples were analyzed for seventeen inorganics, organochlorine

Table 2-2
Analyses of Soil and Groundwater Samples
from the CANS C-2 Area

Chemical	Soil (0.5 ft deep) Freq. of Detection	Geometric Mean (mg/kg)	Maximum Concen. (mg/kg)	Maximum Concen. Location	Soil Core WA-5 (6.0-6.5 ft deep) Conc. (mg/kg)	Ground-Water Well WA-6 Conc. (ug/l)
Inorganics:						
Antimony	0/10	-	-	-	-	-
Arsenic	0/10	-	-	-	-	-
Barium	10/10	37	53	CN-8,10	21	-
Beryllium	0/10	-	-	-	-	-
Cadmium	10/10	5.6	8.7	CN-6	0.55	-
Chromium	10/10	27	44	CN-3,10	23	130
Cobalt	10/10	4.5	6	CN-5	3.6	-
Copper	10/10	12	26	CN-3	23	-
Lead	10/10	38	120	CN-6	-	-
Mercury	2/10	0.16	0.2	CN-4	-	-
Molybdenum	0/10	-	-	-	-	-
Nickel	10/10	25	36	CN-6	19	-
Selenium	0/10	-	-	-	-	-
Silver	0/10	-	-	-	-	-
Thallium	0/10	-	-	-	-	-
Vanadium	10/10	15	24	CN-1	15	-
Zinc	10/10		190	CN-10	18	-
Organics:						
2,4-D	0/10	-	-	-	-	2
Petroleum Hydrocarbons	1/10	-	0.05	CN-5	-	-

Data from Wahler/WESTDIV (1985).
Geometric means of detected samples only.
PCBs and chlorinated pesticides by EPA Method 608 were not detected;
Detection limits were as follows: PCBs, 0.5 mg/kg (soils) and 0.1 ug/l (water);
chlorinated pesticides, 0.02 mg/kg (soils) and 0.2 to 0.05 ug/l (water),
except methoxychlor (0.5 mg/kg in soils), and toxaphene (2 mg/kg in soils
and 1 ug/l in water).
Detection limits for 2,4-D and 2,4,5-TP (chlorinated herbicides) were 1 ug/kg for
soils and 0.1 ug/l for water.
Detection limits for petroleum hydrocarbons were 0.05 mg/kg in shallow soils, 5 mg/kg
in the deep soil sample, and 1 mg/l in the water sample.
Dissolved metals only analyzed in groundwater sample from WA-6.
Soil data reported on a moist-sample-weight (as received) basis.

pesticides, and PCBs. Table 2-3 presents frequencies of detection, geometric means, maximum values and sample location of maximum values of each detected chemical. Sample locations are shown in Figure 2-6. Two shallow sediment samples collected in the channel of the Turning Basin southwest of the Seaplane Lagoon were used as background samples by Wahler/WESTDIV (1985). Twelve of the seventeen inorganics were detected. The geometric mean concentrations of the detected samples can be compared to the geometric mean concentrations of the two background samples for each chemical. Sample mean concentrations exceed mean background levels for the following chemicals: barium (1.3x), chromium (1.7x), cobalt (1.1x), copper (1.8x), lead (3.2x), mercury (1.5x), nickel (1.2x), selenium (1.1x), and zinc (1.7x). Cadmium, arsenic, and thallium were not detected in the background samples. The concentrations of these compounds can instead be compared to the detection limits reported for the background samples. The geometric mean of cadmium in the detected samples exceeds three times the detection limit measured in the background samples for cadmium. Single hits of arsenic and thallium were 1.9 times and 2.3 times, respectively, above the detection limit reported for the background sample. Organochlorine pesticides and PCBs were not above the quantification level in any sample analyzed.

2.3.17 Estuary (Oakland Inner Harbor)

Sediment core samples (5 to 26 feet in length) were collected from three reaches within the Oakland Inner Harbor and from two reaches within Oakland Outer Harbor in December 1986 (U.S. Corps of Engineers, 1988). A reach is an arm of the sea extending into land. The samples were screened for the presence of selected metals, oil and grease, petroleum hydrocarbons, organochlorine pesticides, and PCBs. Table 2-4 presents frequencies of detection, geometric means, maximum values and sample location of maximum values of each detected chemical. Core sample locations are shown in Figures 2-7, 2-8, and 2-9. The geometric mean of the two shallow sediment background samples collected from the channel south of the Seaplane Lagoon is included in Table 2-4 (Wahler/WESTDIV, 1985). These background samples generally had

Table 2-3
Analyses of Sediment Samples
from the Seaplane Lagoon

Chemical	No. of Detects	No. of Samples	Geometric Mean (mg/kg)	Maximum Concen. (mg/kg)	Max. Conc. Location Well No.	Detection Limits (mg/kg)
Antimony	0	8	-	-	-	3
Arsenic	1	8	-	5.7	SBA-5	3
Barium	8	8	25	29	SBA-4	
Beryllium	0	8	-	-	-	0.5
Cadmium	7	8	1.5	6.2	SBA-4	0.5
Chromium	8	8	50	72	SBA-4	
Cobalt	8	8	6.1	6.5	SBA-5	
Copper	8	8	33	50	SBA-4	
Lead	8	8	31	110	SBA-4	
Mercury	8	8	0.18	0.39	SBA-4	0.1
Molybdenum	0	8	-	-	-	10
Nickel	7	8	34	37	SBA-8	
Selenium	7	8	9	11	SBA-5	
Silver	0	8	-	-	-	2
Thallium	1	8	-	6.9	SBA-5	3
Vanadium	0	8	-	-	-	
Zinc	8	8	71	110	SBA-4	

Data taken from Wahler/WESTDIV (1985).
Data reported on a wet-sample-weight (as received basis).
Geometric means of detected samples only.
"-" = none detected
PCBs (EPA Method 608) less than the detection limit of 0.3 mg/kg.
Organochlorine pesticides not detected.

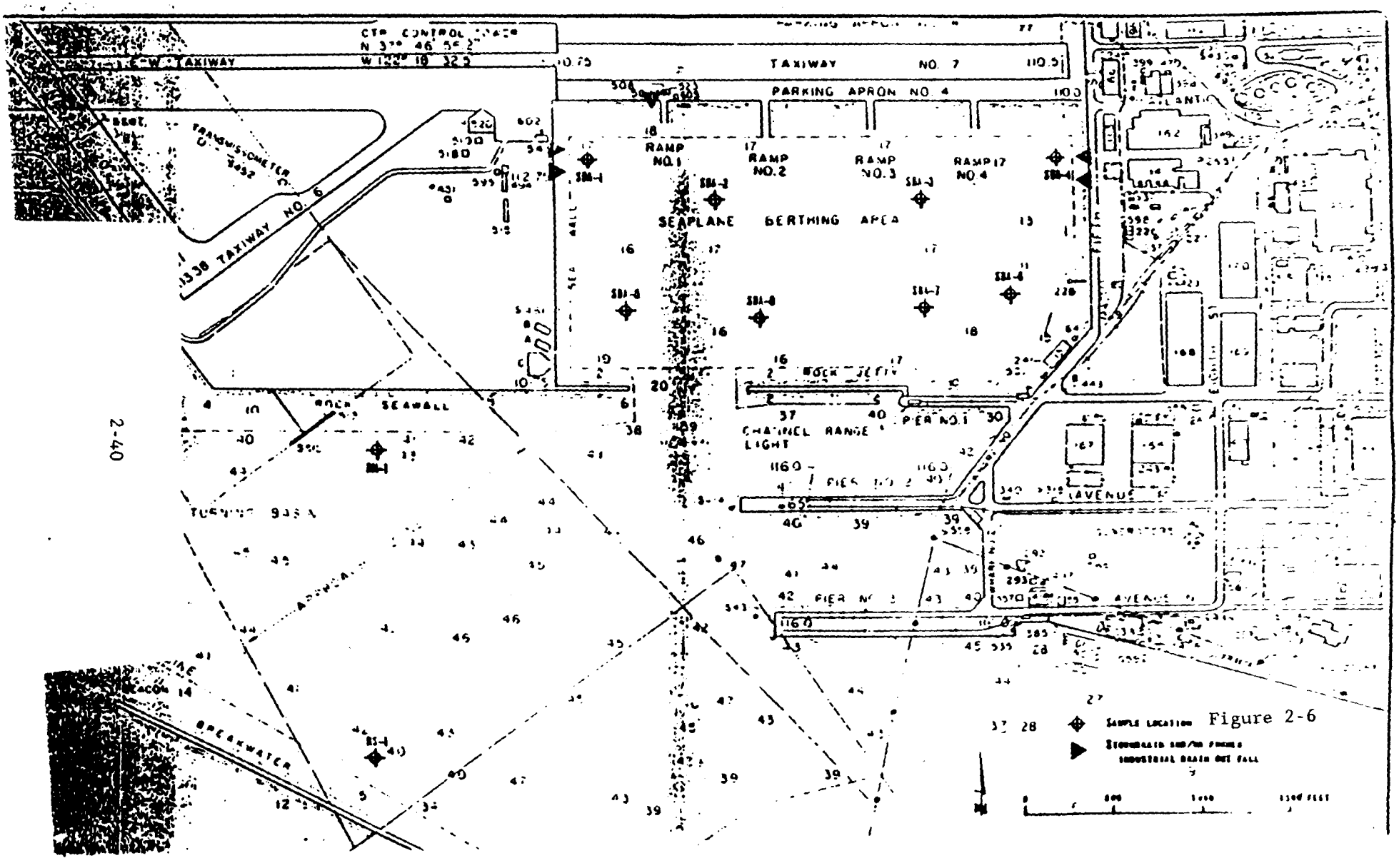

TAXIWAY NO. 7
PARKING APRON NO. 4
RAMP NO.1
RAMP NO.2
RAMP NO.3
RAMP NO.4
SEAPLANE BERTHING AREA
TAXIWAY NO. 6
ROCK SEAWALL
CHANNEL RANGE LIGHT
PIER NO.1
PIER NO.2
AVENUE
BREAKWATER
SAMPLE LOCATION

Figure 2-6

Table 2-4
Analyses of Sediment Samples from Oakland Harbor

Chemical	Oakland Inner Harbor				Oakland Outer Harbor				Potential backgrounds
	Freq. of Detection	Geometric Mean (mg/kg)	Maximum Concen. (mg/kg)	Max. Conc Location Well No.	Freq. of Detection	Geometric Mean (mg/kg)	Maximum Concen. (mg/kg)	Max. Conc Location Well No.	Geometric Mean (mg/kg)
Inorganics:									
Arsenic	4/8	6	20	3dd	3/3	9	9	1bb,1cc	-
Cadmium	8/8	0.42	2.7	3dd	3/3	0.47	0.94	1bb	-
Chromium	8/8	66	130	3dd	3/3	65	86	1ee	29
Copper	8/8	47	440	3dd	3/3	38	60	1ee	18
Lead	8/8	22	200	3dd	3/3	19	35	1bb	9.8
Mercury	8/8	0.39	3.4	3dd	3/3	0.27	0.38	1bb	0.12
Nickel	4/8	61	98	3dd	3/3	61	90	1ee	28
Selenium	0/8	-	-	-	0/3	-	-	-	8
Silver	3/8	0.1	0.2	2cc	2/3	0.14	0.2	1ee	-
Zinc	8/8	106	540	3dd	3/3	101	163	1ee	43
Organics:									
Oil and Grease	8/8	640	3600	3dd	3/3	462	875	1ee	na
Petroleum Hydrocarbons	8/8	97	205	3aa	3/3	117	200	1bb	na

Background sediment data taken from Wahler/WESTDIV (1985).
Sediment data from Oakland Harbor taken from U.S. Army Corps of Engineers (1988).
Data reported on a wet-sample-weight (as received basis)
Geometric means of detected samples only.
"-" = none detected. Detection limits of background samples are as follows: Sb, Se, As (3 mg/kg); and Cd (0.5 mg/kg).
Detection limit of Se in the Oakland Harbor samples was 3 mg/kg.
PCBs were not detected in any of the samples above the detection limit of 3 ug/kg.
Organochlorine pesticides were not detected in any samples above the detection limits (0.5 to 10 ug/kg).
na = not analyzed.

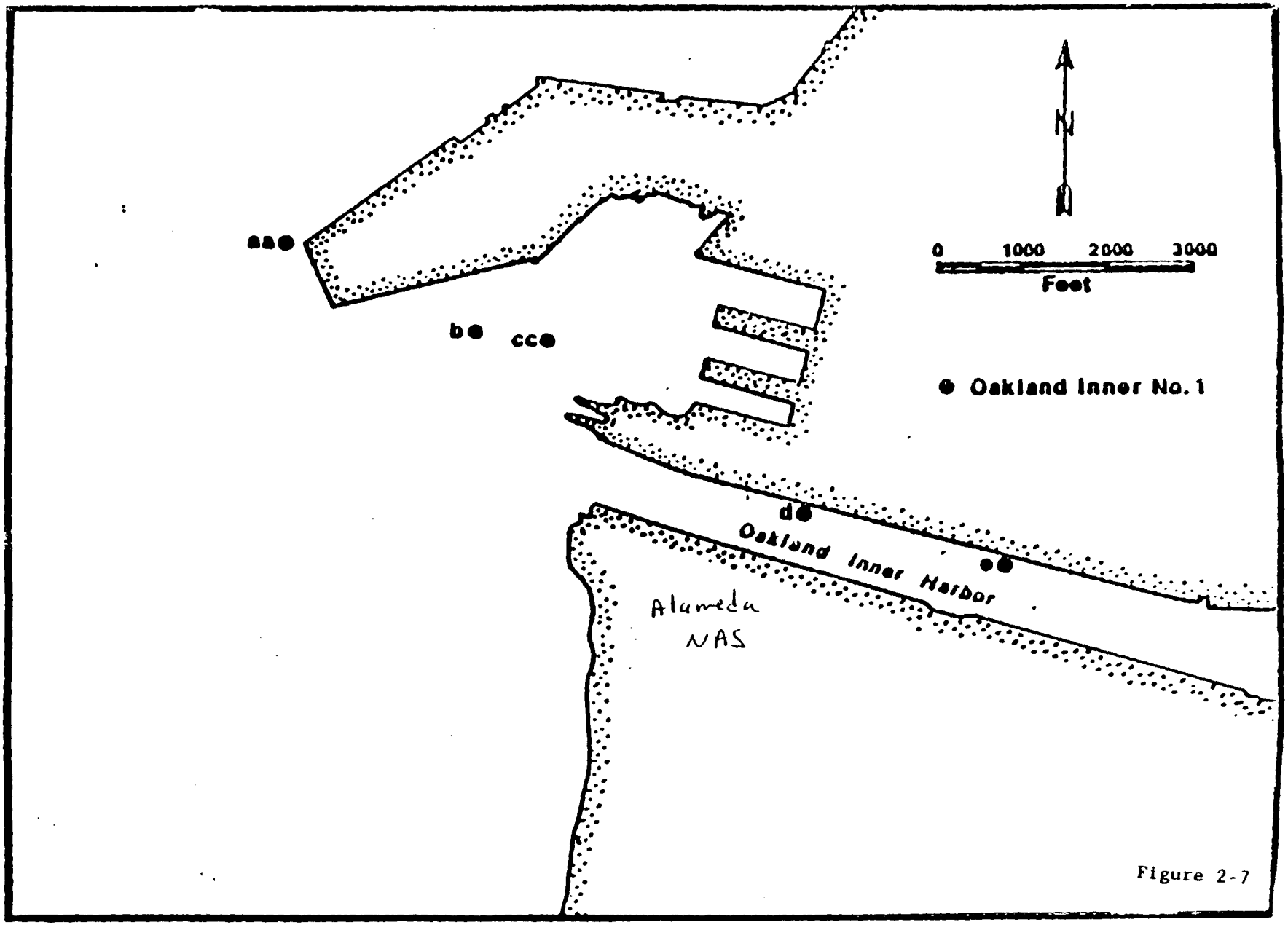

N
0 1000 2000 3000
Feet
a
b
c
d
Oakland Inner No. 1
Oakland Inner Harbor
Alameda
NAS

Figure 2-7

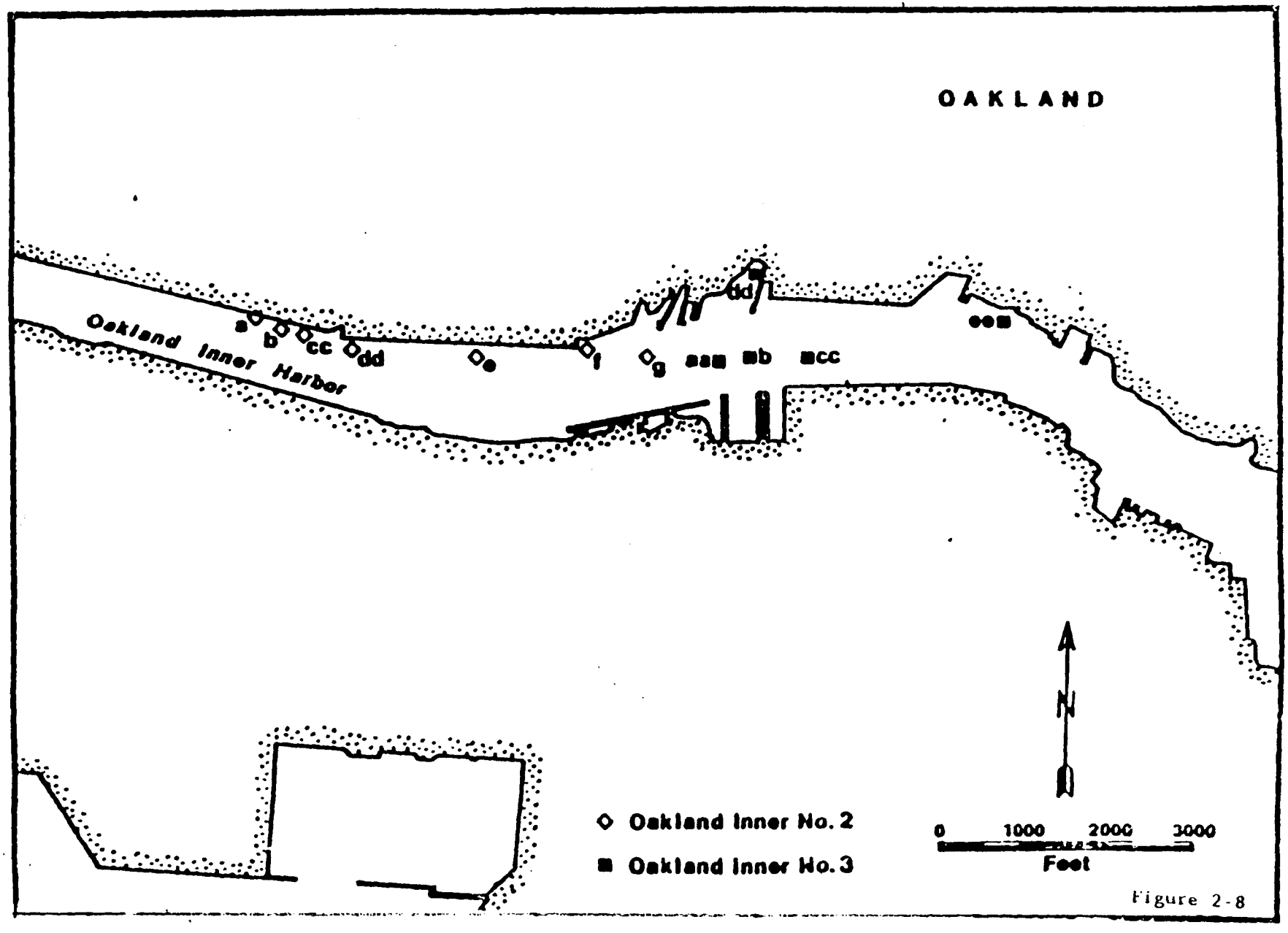

OAKLAND
Oakland Inner Harbor
a
b
cc
dd
e
f
g
aa
bb
cc
dd
ee
N
Oakland Inner No. 2
Oakland Inner No. 3
0
1000
2000
3000
Feet

Figure 2-8

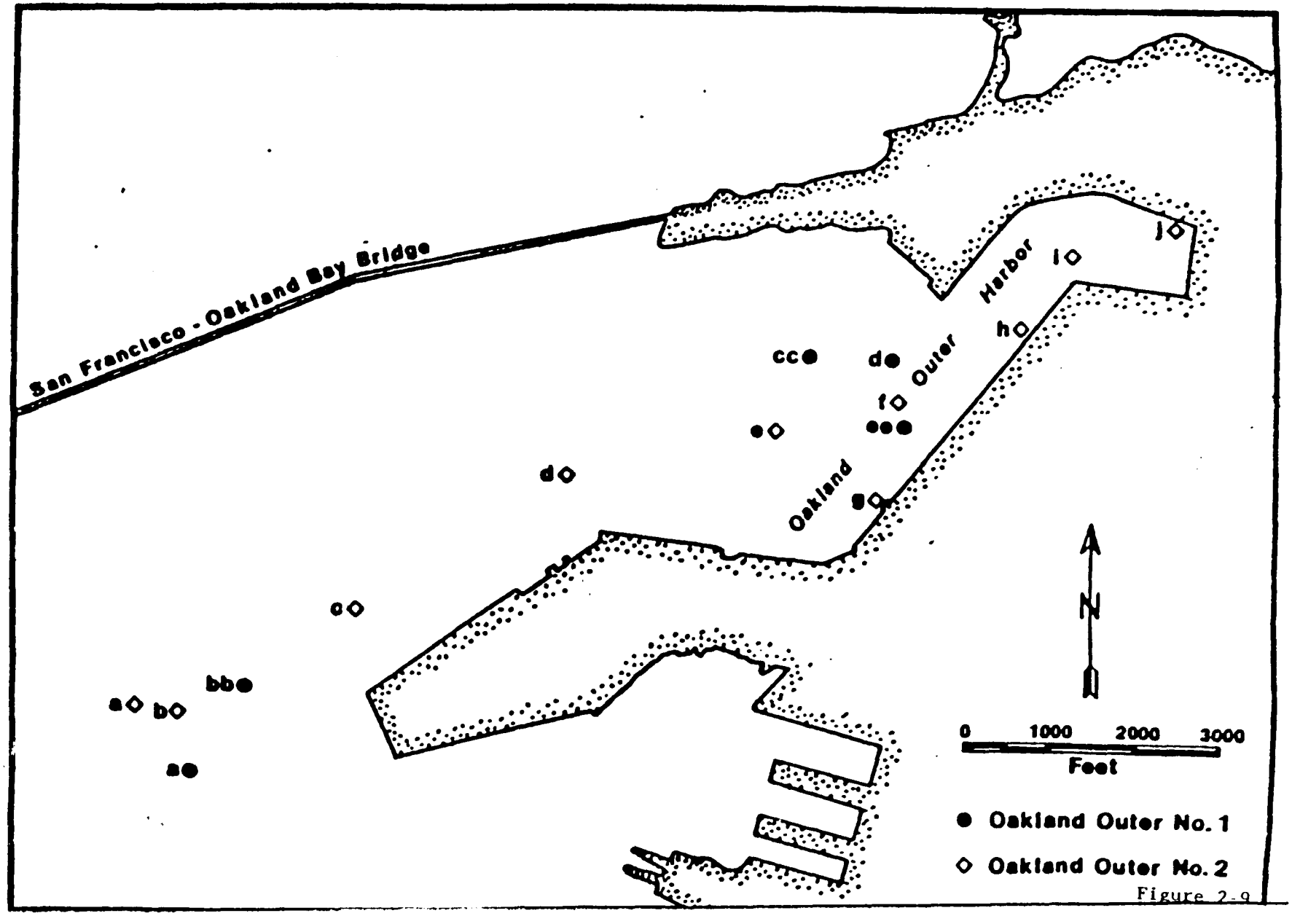

San Francisco - Oakland Bay Bridge
Harbor
Outer
Oakland
cc
d
f
h
i
j
g
d
c
a
b
bb
N
0 1000 2000 3000
Feet
Oakland Outer No. 1
Oakland Outer No. 2

Figure 2-9

higher detection limits than the Oakland Harbor samples. In the final PHEE only background samples representative of the estuary will be used.

The geometric mean concentrations of sediment samples from the Oakland Inner Harbor exceeded the mean background sediment samples for the following chemicals: chromium (2.3x), copper (2.6x), lead (2.3x), mercury (3.3x), nickel (2.2x), and zinc (2.5x). Arsenic (6 mg/kg), cadmium (0.42 mg/kg), and silver (0.1 mg/kg) were detected in the Oakland Inner Harbor sediments, but not in the background sediments. The geometric mean concentrations of sediment samples from the Oakland Outer Harbor exceeded the geometric mean of the background sediment samples for the following chemicals: chromium (2.2x), copper (2.1x), lead (1.9x), mercury (2.3x), nickel (2.2x), and zinc (2.4x). Arsenic (9 mg/kg), cadmium (0.47 mg/kg), and silver (0.14 mg/kg) were detected in the Oakland Outer Harbor sediments, but not in the background sediments samples which generally had higher detection limits than the Oakland Harbor samples. Oil and grease and total petroleum hydrocarbons were present in all samples collected in the Oakland Harbor, but were not analyzed in the background sediment samples. The geometric mean concentrations of oil and grease in the sediment samples were 640 mg/kg in the Oakland Inner Harbor, and 462 mg/kg in the Oakland Outer Harbor. The geometric mean concentrations of petroleum hydrocarbons in the sediment samples were 97 mg/kg in the Oakland Inner Harbor and 117 mg/kg in the Oakland Outer Harbor. PCBs and organochlorine pesticides were not detected in any of the 11 sediment samples collected from Oakland Harbor (U.S. Corps of Engineers, 1988).

2.3.18 1943-1956 Disposal Area

Five monitoring wells were installed within the boundaries of the 1943-1956 Disposal Area in October 1984 by Wahler Associates during the Verification Study of the NAS-Alameda. Soil core samples and groundwater samples were collected from each monitoring well. The locations of the five monitoring wells are shown in Figure 2-10.

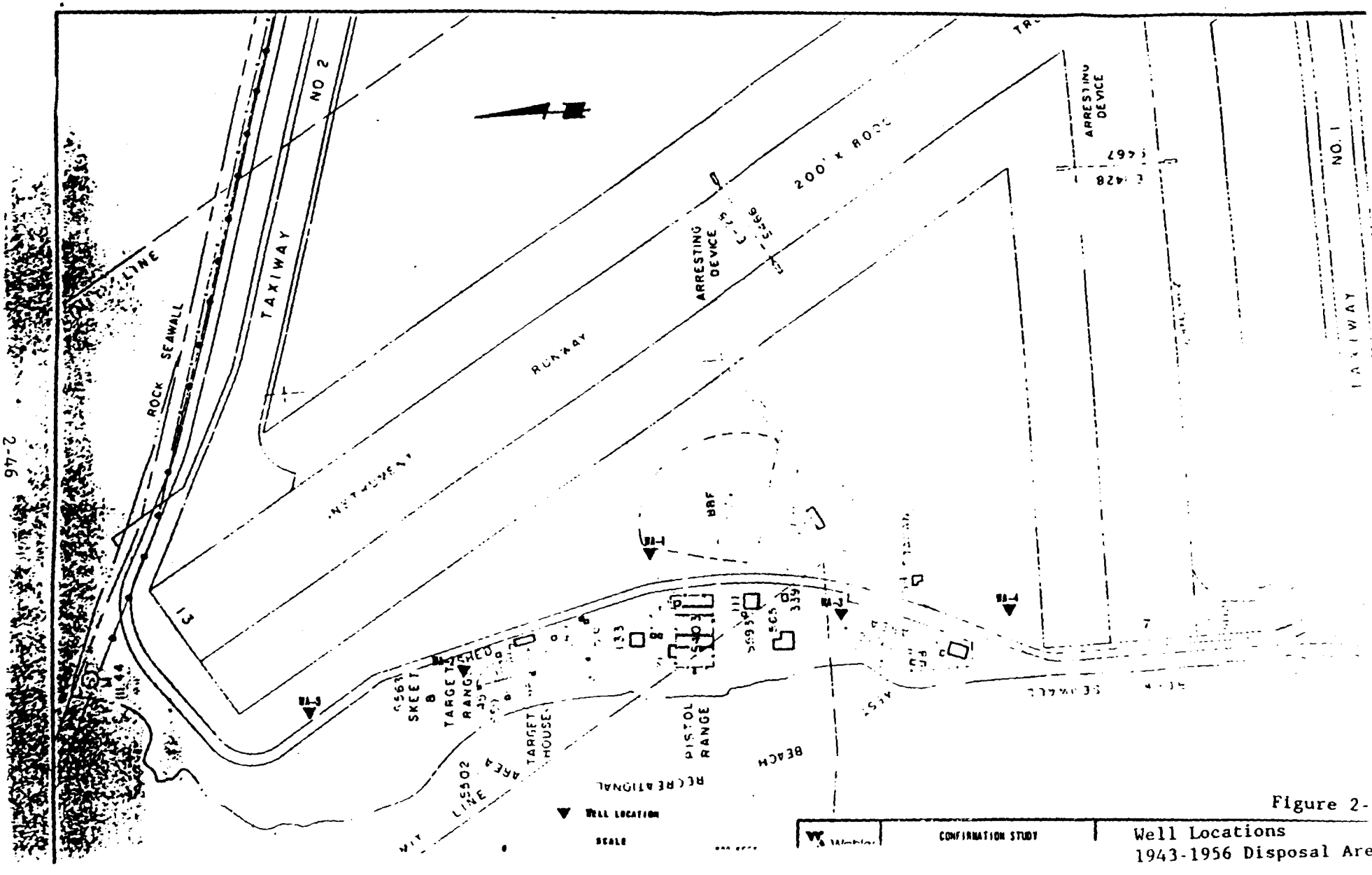


Figure 2-10
Well Locations
1943-1956 Disposal Area

Soils: Core samples collected about six feet below the ground surface during installation of each of the five monitoring wells were analyzed for the presence of metals, purgeable organics, base/neutral and acid extractable organics, and radiation. Table 2-5 presents frequencies of detection, geometric means, maximum values, and well location of detected values of each detected chemical. No background soil samples were reported by Wahler/WESTDIV (1985). The concentrations of gross alpha and gross beta radiation in the soil cores ranged from 0.1 ±4.8 pCi/g to 45.7 ±10.8 pCi/g and from 10.5 ±3.3 pCi/g to 31.4 ±4.4 pCi/g, respectively. The pH of core samples ranged from 7.9 to 8.8 (Wahler/WESTDIV, 1985).

Inorganics detected in the soil core samples included arsenic, barium, cadmium, chromium, cobalt, copper, lead, mercury, nickel, vanadium, and zinc. Wahler/WESTDIV (1985) reported elevated levels of copper, lead, and zinc, although an analysis of a background soil sample for comparison was not included in the report. The following polycyclic aromatic hydrocarbons were detected in the core soil sample collected from well WA-3: acenaphthene, acenaphthylene, naphthalene, benzo(a)anthracene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(g,h,i)perylene, benzo(a)pyrene, indeno(1,2,3-c,d)pyrene, chrysene, fluorene, phenanthrene, dibenzofuran, and 2-methylnaphthalene. In addition, bis(2-ethylhexyl)phthalate, di-n-butyl phthalate and acetone were each detected in one of the five soil cores. Because of the absence of background data, the concentration of detected chemicals will not be further analyzed in the preliminary PHEE. Wahler/WESTDIV (1985) reported that the gross alpha and gross beta radiation levels are similar to levels found in off-site soils in the Bay area.

Groundwater: Groundwater samples were collected from the five monitoring wells in January 1985. Monitoring wells were screened from 5 feet to 17 or 25 feet below the surface level. Groundwater samples were analyzed for the presence of metals, purgeable organics, base/neutral and acid extractable organics, and radiation. Table 2-5 presents frequencies of detection, geometric means, maximum values, and well location of maximum values of each detected chemical. No background groundwater samples were reported to be

Table 2-5 : 1943-1956 Disposal Are Disposal Area, Soil and Groundwater Data

	Analyses of 1984 soil samples (6-6.5 ft. deep)				Analyses of 1985 groundwater samples			
Chemical	Freq. of Detection	Geometric Mean (mg/kg)	Maximum Concen. (mg/kg)	Max. Conc. Location Well No.	Freq. of Detection	Geometric Mean (mg/l)	Maximum Concen. (mg/l)	Max. Conc. Location Well No.
Inorganics:								
Antimony	0/5	-	-	-	0/5	-	-	-
Arsenic	2/5	7	9.1	WA-5	0/5	-	-	-
Barium	5/5	59	250	WA-3	0/5	-	-	-
Beryllium	0/5	-	-	-	0/5	-	-	-
Cadmium	5/5	3.4	24	WA-1	0/5	-	-	-
Chromium	5/5	43	90	WA-1	0/5	-	-	-
Cobalt	5/5	5.9	9.4	WA-5	0/5	-	-	-
Copper	5/5	59	330	WA-3	0/5	-	-	-
Lead	5/5	117	1100	WA-1	0/5	-	-	-
Mercury	5/5	0.3	2.3	WA-3	0/5	-	-	-
Molybdenum	0/5	-	-	-	1/5	-	0.77	WA-3
Nickel	5/5	42	70	WA-1	0/5	-	-	-
Selenium	0/5	-	-	-	0/5	-	-	-
Silver	0/5	-	-	-	0/5	-	-	-
Thallium	0/5	-	-	-	0/5	-	-	-
Vanadium	5/5	15	22	WA-2	0/5	-	-	-
Zinc	5/5	131	1800	WA-3	1/5	-	0.13	WA-1
Organics:								
1,1,1-Trichloroethane	0/5	-	-	-	2/5	0.038	0.291	WA-1
trans-1,2-dichloroethylene	0/5	-	-	-	3/5	0.123	0.957	WA-1
Benzene	0/5	-	-	-	1/5	-	0.009	WA-5
Acetone	1/5	-	0.058	WA-1	0/5	-	-	-
Bis(2-ethylhexyl)-phthalate	1/5	-	0.625	WA-2	1/5	-	0.06	WA-1
Di-n-butyl phthalate	3/5	1.3	2.7	WA-1	0/5	-	-	-
Acenaphthene	1/5	-	2	WA-3	1/5	-	0.064	WA-2
Acenaphthylene	0/5	-	-	-	1/5	-	0.005	WA-2
Naphthalene	1/5	-	5.2	WA-3	0/5	-	-	-
Benzo(a)anthracene	1/5	-	0.37	WA-3	0/5	-	-	-
Benzo(b)fluoranthene	1/5	-	0.58	WA-3	0/5	-	-	-
Benzo(ghi)perylene	1/5	-	0.44	WA-3	0/5	-	-	-
Benzo(a)pyrene	1/5	-	1.33	WA-3	0/5	-	-	-
Indeno(1,2,3-cd)pyrene	1/5	-	1	WA-3	0/5	-	-	-
Pyrene	0/5	-	-	-	1/5	-	0.043	WA-2
Chrysene	1/5	-	0.47	WA-3	0/5	-	-	-
Fluorene	1/5	-	1.84	WA-3	1/5	-	0.016	WA-2
Phenanthrene	1/5	-	0.2	WA-3	0/5	-	-	-
Dibenzofuran	1/5	-	1.36	WA-3	1/5	-	1.014	WA-2
2-methylnaphthalene	1/5	-	0.8	WA-3	0/5	-	-	-
2-cyclohexen-1-one*	0/5	-	-	-	1/5	-	0.01	WA-3
2,5-diethyltetrahydrofuran*	0/5	-	-	-	1/5	-	0.043	WA-3

Data taken from Wahler/WESTDIV (1985)

* Estimated concentrations, tentative identification.

Inorganic analytical detection limits generally were 1 to 5 mg/kg for soil & 0.1 to 1 mg/l for groundwater samples.

Organic analytical detection limits generally were 0.001 to 0.04 mg/kg for soil & 0.001 mg/l for groundwater samples.

Soil data reported on a moist-sample-weight (as received) basis.

Geometric means of detected values only. "-" = Not detected.

collected or analyzed by Wahler/WESTDIV (1985). The concentrations of gross alpha and gross beta radiation in the groundwater samples ranged from 0.4 ±2.8 pCi/g to 7.2 ±6.2 pCi/g and from 33.8 ±57.4 pCi/g to 69.3 ±31.6 pCi/g, respectively. Wahler/WESTDIV (1985) reported that high levels of suspended solids in the groundwater samples interfered with the analytical method used to measure radiation. The pH of groundwater samples ranged from 6.7 to 7.6. The electrical conductivity in groundwater samples ranged from 750 to 11,800 umhos/cm (Wahler/WESTDIV, 1985).

The dissolved inorganics, detected in one groundwater sample each, were molybdenum and zinc. Organic contaminants detected in two groundwater samples each were 1,1,1-trichloroethane and 1,2-dichloroethylene. Organic contaminants detected in one of the five groundwater samples were bis(2-ethylhexyl)phthalate, acenaphthene, acenaphthylene, pyrene, fluorene, and dibenzofuran. Tentatively identified compounds in one groundwater sample included 2-cyclohexen-1-one and 2,5-diethyltetrahydrofuran. The concentrations of tentatively identified compounds are only qualitative.

2.3.19 West Beach Landfill

Groundwater: In 1976, nineteen monitoring wells were installed by Harding Lawson Associates/WESTDIV (HLA/WESTDIV). An additional six monitoring wells were installed by HLA/WESTDIV in July 1983. All monitoring wells are located within the boundaries of the landfill area, and the locations are shown in Figure 2-11. The screened interval of the monitoring wells extends 20 to 35 feet deep with the top of the screen located two to seven feet below the ground surface. HLA/WESTDIV (1978) reported that the direction of groundwater flow is generally toward the Bay, but the hydraulic gradient is small, particularly in the western portion of the landfill.

Seven groundwater sampling rounds of selected wells are reported: November 1976; March, July and October of 1977; and March, early August, and middle August 1983. A total of 32 samples from nineteen monitoring wells were collected in 1976 and 1977, and analyzed for water quality parameters

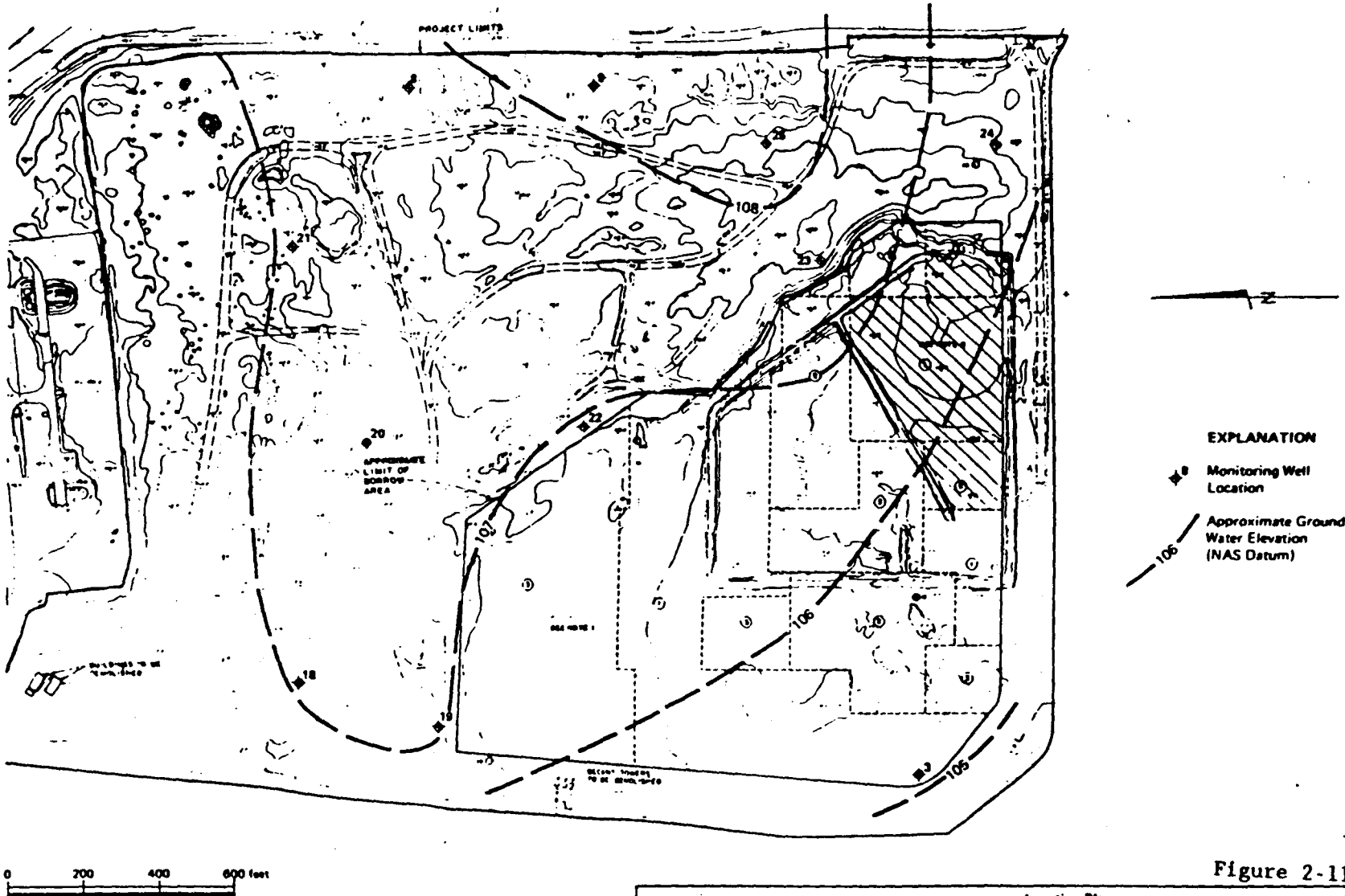


Figure 2-11

Harding Lawson Associates Engineers, Geologists & Geophysicists	Location Plan Sanitary Landfill Site Alameda Naval Air Station Alameda, California				PLATE 1
DRAWN MLO	JOB NUMBER 2176,059.01	APPROVED	DATE 10/83	REVISED	DATE

including metals, PCBs, and phenol. Table 2-6 presents frequencies of detection, geometric means, maximum values, and well location of maximum values of each detected chemical. In addition, potential background samples were collected in 1977 from observation wells #10 and #20. Samples of Bay water were taken at high and low tides south of the landfill in April and July 1977 to provide additional background references and to detect saline intrusion. Analytical data for chemicals in the background samples and the surface water sample from the Bay are shown in Table 2-7. Observation well #20 was located east of the landfill and was considered unaffected by the waste disposal practices at the landfill. Although east of the landfill, observation well #10 is closer to the landfill and during periods of high rainfall the hydraulic gradient may be locally reversed from the landfill toward observation well #10 (HLA/WESTDIV, 1978).

Elevated levels of oil and grease, sulfide, iron, nitrate nitrogen, mercury, lead, total phosphate, total Kjeldahl nitrogen, total chromium, and turbidity were reported by HLA/WESTDIV (1978).

Eight groundwater samples were collected in March 1983 from eight monitoring wells installed in 1976 and were analyzed for EPA inorganic and organic priority pollutants. Trace amounts of phenol and polychlorinated biphenyls were reported. Groundwater samples collected in early August 1983 from three of the monitoring wells installed in July 1983 were analyzed for organic priority pollutants and dissolved metals. An additional eleven samples were reported to have been submitted for chemical analyses by HLA, but these analytical results are not included in HLA's report. Eleven groundwater samples collected by HLA in mid August 1983 were analyzed for organic priority pollutants and pesticides by more sensitive analytical methods than the previously submitted groundwater samples. HLA/WESTDIV (1983) reported that PCBs (< 10 $\mu g/L$) were the only contaminants detected in the last round of sampling. No background groundwater samples were reported to be collected or analyzed by HLA in the 1983 sampling rounds. The concentrations of chromium and lead measured in the background samples collected from observations wells #10 and #20 exceed the geometric mean concentrations of the groundwater

Table 2-6
Groundwater Analyses of Samples
from the Sanitary Landfill

Chemical	No. of Detects	No. of Samples	Geometric Mean (ug/L)	Maximum Concen. (ug/L)	Max. Conc. Location Well No.
Inorganics:					
Antimony	2	3	659	700	23
Arsenic	11	11	43	90	17
Beryllium	1	11	-	12	17
Cadmium	38	43	32	460	5
Chromium	27	43	104	1000	4
Copper	11	11	63	720	17
Lead	41	43	186	650	4
Magnesium	40	40	270000	950000	2
Mercury	11	43	1.3	3.4	2
Nickel	11	11	120	400	23
Selenium	10	11	42	80	17
Thallium	2	3	0.2	0.2	22,23
Zinc	11	11	64	480	17
Organics:					
Oil & Grease	40	40	8100	80000	9
TICH	7	8	0.19	0.6	19
Benzene	1	22	-	6	21
Chlorobenzene	1	22	-	31	21
Ethylbenzene	1	22	-	5	21
Toluene	1	22	-	235	23
Acetone	1	22	-	620	21
o-xylene	1	22	-	11	21
Phenol	6	22	12	26	17
2,4-dimethylphenol	1	22	-	38	23
2-methylnaphthalene	1	22	-	16	21
naphthalene	2	22	92	104	21
Bis(2-ethylhexyl)-phthalate	1	22	-	10	21
D-BHC	1	22	-	0.2	23
pp-DDT (4,4')	1	22	-	0.7	23
Endrin Aldehyde	1	22	-	0.1	23
Endosulfan Sulfate	1	22	0.2	0.5	23
a-BHC	1	22	-	0.2	21
Heptachlor	2	22	0.3	0.4	21
g-BHC (lindane)	1	22	-	0.3	22
Aldrin	1	22	-	0.1	22
PCBs	2	14	5.6	8	20

Data from HLA/WESTDIV (1978,1983).
Geometric means of detected samples only.
Detection limits are as follows: 32 1977 samples: Hg, 1 ug/l; Pb, 40 ug/l; Total Cr, 40 ug/l; Cd, 10 ug/l; 1983 data: volatile organics, 1 to 10 ug/l; acid extractables, 0.1 to 10 ug/l; base/neutral extractables, less than 2 ug/l; pesticides, 0.1 to 1 ug/l.
TICH = Total identifable chlorinated hydrocarbon fraction (as arochlor 1248).
Dissolved metals only analyzed.

Table 2-7
Potential background concentrations for
Sanitary Landfill groundwater (a)

	Bay Averages (b)		Well #10	Well #20 (c)
Date:	March 1977	July 1977	October 1977	October 1977
Chemical	(mg/l)	(mg/l)	(mg/l)	(mg/l)
Inorganics:				
Iron	0.97	0.11	470	330
Magnesium	1100	1200	32	30
Potassium	415	270	1.4	1.6
Sodium	9600	3490	65	50
Mercury	-	-	0.0082	0.001
Lead	0.35	-	0.51	0.32
Chromium	0.04	1.03	1.56	0.9
Cadmium	0.033	-	0.34	-
Organics:				
Oil & Grease	6.45	0.73	12	8.8

a Data taken from HLA/WESTDIV (1978).
b Each bay average is an arithmetic average of a low tide and a high tide concentration as reported by HLA/WESTDIV (1978). Bay water collected off southern boundary of landfill.
c Observation wells located east of landfill
"-" = not detected, detection limits were 0.001 mg/l for Hg, 0.04 mg/l for Pb, and 0.01 mg/l for Cd.

samples collected from within the boundary of the landfill. The mean concentrations of mercury and cadmium in the groundwater samples from the landfill are about the same as the concentrations found in the background groundwater samples.

2.3.20 Yard D-13

Environmental sampling results are not available for this area.

2.4 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

In this section chemicals of potential concern are identified for each of the twenty study areas of interest, by media. These chemicals are identified based on the available data from previous sampling rounds presented in Section 2.3, and the chemicals identified in Section 2.1 as being related to present or past site activities. The purpose of generating this initial list of chemicals of potential concern is to focus sampling efforts in the RI. This list must be considered tentative because of the paucity of currently available data, the undocumented QA/QC procedures used to date and, in general, the lack of background concentration data. Chemicals of potential concern are identified for soils and groundwater for all study areas except for the Seaplane Lagoon and Oakland Inner Harbor. Chemicals of potential concern for sediments and surface waters are identified for these two areas. Chemicals of potential concern are not identified for air because of the lack of data on any stack emissions.

When a large number of chemicals is detected at a site, a subset of chemicals of concern is generally selected in order to focus the risk assessment on those contaminants that are most likely to pose risks at the site. When the final RI data are available, a final chemical selection process will be used for each study area by media. This process usually involves consideration of the following factors: historical use on the site; comparison of sample concentrations with concentrations in field and laboratory blanks; comparison of sample concentrations with background

concentrations; the frequency a chemical is detected; and the toxilogical and physicochemical properties of the chemical.

The potential chemicals of concern for each of the 20 study areas of interest are discussed below. Table 2-8 summarizes the currently available sampling data from each study area. Table 2-9 summarizes the chemicals detected in measurable concentrations in each study area for which sampling results are available. The potential chemicals of concern are identified on the basis of their presence at the study area due to known site activities. For the seven study areas where sampling data are available, gaps in the current database are identified. These chemicals are of potential concern as soil contaminants. Since they may also leach from soils into the underlying groundwater, they are of concern as potential groundwater contaminants.

2.4.1 Building 41

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams at Building 41 and are chemicals of potential concern:

- Chlorinated organic solvents from PD 680 dry cleaner,
- Trichlorotrifluoroethane and 1,1,1-trichloroethane solvents from cleaning solvents,
- Metals, organic solvents (e.g., methyl ethyl ketone) from paint wastes,
- Xylene, toluene, phenol and other organic solvents from paint strippers, and
- Metals, PCBs, PAHs and petroleum hydrocarbons from oil and hydraulic fluid wastes.2.4.2 Buildings 162, 459, and 547 (Service Stations).

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams and are chemicals of potential concern:

Table 2-8

Chemicals of Potential Concern Analyzed
in Soils and Groundwater from each Study Area

Study Area	Metals[a]	Cyanide	VOCs[b]	BNA Extractables[c]	Petroleum Hydrocarbons	PCBs[d]	Pesticides[e]	Radiation[f]	Asbestos
Building 41									
Buildings 162, 459, and 547									
Building 10									
Area 97	X				X				
Oil Refinery									
Fire Training Area									
Building 114									
Building 5									
Building 360		X	X						
Building 410									
Buildings 400 and 530									
Building 14									

Table 2-8
(Continued)

Study Area	Metals[a]	Cyanide	VOCs[b]	BNA Extractables[c]	Petroleum Hydrocarbons	PCBs[d]	Pesticides[e]	Radiation[f]	Asbestos
Buildings 301 and 389						X			
CANs C-2 Area	X				X	X	X		
Sewer System									
1943-1956 Disposal Area	X		X	X	X			X	
Seaplane Lagoon	X					X	X		
Estuary	X				X	X	X		
West Beach Landfill	X		X	X	X	X		X	
Yard D-13									

a Metals include antimony, arsenic, beryllium, cadmium, chromium, copper, lead, mercury, nickel, selenium, silver, thallium and zinc. All metals were not analyzed in each study area.
b Volatile organic compounds include chlorinated hydrocarbons and monocyclic aromatics.
c Base/neutral and acid extractable compounds include phenol derivatives, organochlorine pesticides, polynuclear aromatic hydrocarbons and some polychlorinated biphenyls.
d Polychlorinated biphenyls
e Organophosphorous, organochlorine, chlorophenoxy pesticides and herbicides. Not all pesticides were analyzed in each study area.
f Gross alpha and beta radiation.

Table 2-9

Chemicals
Detected in Media for Study Areas
with Chemical Data

Study Area	Soil[a]/Sediment	Groundwater[b]	Chemical
Area 97	--	X	Lead
	sub	X	Petroleum hydrocarbons
	na	X	Oil and grease
Building 360	sub	na	Cyanide
	sub	na	1,1,1-trichloroethane
Buildings 301 and 389	sfc,sub	na	PCBs
Cans C-2 Area	sfc,sub	--	Barium
	sfc,sub	--	Cadmium
	sfc,sub	X	Chromium
	sfc,sub	--	Cobalt
	sfc,sub	--	Copper
	sfc	--	Lead
	sfc	--	Mercury
	sfc,sub	--	Nickel
	sfc,sub	--	Vanadium
	sfc,sub	--	Zinc
	--	X	2,4-D
	--	X	Endrin
	--	X	1,2-dichloroethylene
	sfc	--	Petroleum hydrocarbons
Seaplane Lagoon[c]	sed	na	Arsenic
	sed	na	Barium
	sed	na	Cadmium
	sed	na	Chromium
	sed	na	Cobalt
	sed	na	Copper
	sed	na	Lead
	sed	na	Mercury
	sed	na	Nickel
	sed	na	Selenium
	sed	na	Thallium
	sed	na	Zinc
Estuary[c]	sed	na	Arsenic
(Oakland Inner Harbor)	sed	na	Cadmium
	sed	na	Chromium
	sed	na	Copper
	sed	na	Lead

Table 2-9
(continued)

Study Area	Soil[a]/Sediment	Groundwater[b]	Chemical
Estuary (continued)	sed	na	Mercury
	sed	na	Nickel
	sed	na	Silver
	sed	na	Zinc
	sed	na	Petroleum hydrocarbons
	sed	na	Oil and grease
1943-1956 Disposal Area	sub	--	Arsenic
	sub	--	Barium
	sub	--	Cadmium
	sub	--	Chromium
	sub	--	Cobalt
	sub	--	Copper
	sub	--	Lead
	sub	--	Mercury
	--	X	Molybdenum
	sub	--	Nickel
	sub	--	Vanadium
	sub	X	Zinc
	--	X	1,1,1-trichloroethane
	--	X	trans-1,2-dichloroethylene
	--	X	Benzene
	sub	--	Acetone
	sub	X	Bis(2-ethylhexyl)phthalate
	sub	--	Di-n-butyl phthalate
	sub	X	Acenaphthene
	--	X	Acenaphthylene
	sub	--	Naphthalene
	sub	--	Benzo(a)anthracene
	sub	--	Benzo(b)fluoranthene
	sub	--	Benzo(g,h,i)perylene
	sub	--	Benzo(a)pyrene
	sub	--	Indeno(1,2,3-cd)-pyrene
	--	X	Pyrene
	sub	--	Chrysene
	sub	X	Fluorene
	sub	--	Phenanthrene
	sub	X	Dibenzofuran
	sub	--	1-methylnaphthalene
	--	X	2-cyclohexen-1-one
	--	X	2,5-diethyltetrahydrofuran
	sub	X	Gross alpha radiation
	sub	X	Gross beta radiation

Table 2-9
(continued)

Study Area	Soil[a]/Sediment	Groundwater[b]	Chemical
West Beach Sanitary Landfill	na	X	Antimony
	na	X	Arsenic
	na	X	Beryllium
	na	X	Cadmium
	na	X	Chromium
	na	X	Copper
	na	X	Lead
	na	X	Magnesium
	na	X	Mercury
	na	X	Nickel
	na	X	Selenium
	na	X	Thallium
	na	X	Zinc
	na	X	Oil and grease
	na	X	Benzene
	na	X	Chlorobenzene
	na	X	Ethylbenzene
	na	X	Toluene
	na	X	Acetone
	na	X	o-xylene
	na	X	Phenol
	na	X	2,4-dimethylphenol
	na	X	2-methylnaphthalenes
	na	X	Naphthalene
	na	X	Bis(2-ethylhexyl)phthalate
	na	X	d-BHC
	na	X	pp-DDT(4,4')
	na	X	Endrin aldehyde
	na	X	Endosulfan sulfate
	na	X	a-BHC
	na	X	Heptachlor
	na	X	g-BHC(Lindane)
	na	X	Aldrin
	na	X	PCBs
	na	X	Petroleum hydrocarbons
	na	X	Gross alpha radiation
	na	X	Gross beta radiation

[a] sub = subsurface soil; sfc = surface soil (0 to 6" deep); na = not analyzed; "--" = not detected

[b] X = detected; "--" = not detected; na = not analyzed

[c] surface water samples not analyzed

- Metals and petroleum hydrocarbons from present and past leaking underground gasoline and waste oil tanks,
- Organic solvents, PCBs and PAHs from present and past leaking underground waste oil tanks,
- Acetone, Freon, and chlorinated hydrocarbon solvents (e.g., 1,1,1-trichloroethane) from waste solvents generated from operations conducted in Building 162, and
- Metals, PCBs, organic solvents, and PAHs from waste lube and hydraulic oils generated from operations conducted in Building 162.

2.4.3 Building 10 (Power Plant)

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams and are chemicals of potential concern:

- Petroleum hydrocarbons and metals from diesel and Bunker C fuels formerly stored in underground storage tanks,
- Morpholine, and caustics in boiler blowdown, and
- Petroleum hydrocarbons, metals, PCBs, and PAHs from waste oil.

2.4.4 Area 97

Available sampling data identified gasoline hydrocarbons and lead in the groundwater (Table 2-9). In addition, benzene, toluene, xylene, and the heavier faction of hydrocarbons should be included in future analyses of groundwater; surface and subsurface grab soil samples should be analyzed for the same chemicals as groundwater. A lower detection limit (e.g., 1 to 5 mg/kg) for gasoline hydrocarbons should be used in future sampling efforts. At least three background samples for each medium should be analyzed for the above suite of chemicals.

2.4.5 Oil Refinery

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams and are chemicals of potential concern:

- Petroleum hydrocarbons (light, heavy, oil and grease), metals, PCBs, PAHs, and organic solvents from asphaltic wastes and stillbottoms buried on site.

Currently available sampling data from Area 97 (Well OW-6) and the Cans C-2 area may detect wastes from the former Oil Refinery disposal area which formerly was located in this area.

2.4.6 Fire Training Area

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams and are chemicals of potential concern:

- Petroleum hydrocarbons, metals, organic solvents, pesticides, PCBs, polychlorinated dibenzodioxins/dibenzofurans, and PAHS from fuel and oil wastes

2.4.7 Building 114

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams and are chemicals of potential concern:

- Pesticides (e.g., Roundup, Princep, Krovar I, Malathion, Diazinon, Warfarin, chlordane, lindane, DDT, Telvar, Chlorvar, 2,4-D),
- Metals, petroleum hydrocarbons, PCBs, and PAHs from oil and grease tank separator,
- Organic solvents (e.g., phenol) from paint strippers, and
- Metals and organic solvents from paint wastes.

2.4.8 Building 5

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams and are chemicals of potential concern:

- Metals (e.g., chromium, nickel, silver, lead, cadmium, zinc, and copper) and cyanide from electroplating bath liquids and sludges
- Acids and bases from electroplating operations,
- Aluminum, iron, and chromium in wastewater from the conversion coating process,
- Phenol, and other organic solvents (e.g., methylene chloride), chromium, PCBs, and petroleum hydrocarbons in wastewaters generated from the paint stripping process,
- Chlorinated hydrocarbon solvents (e.g., 1,1,1-trichloroethane, carbon tetrachloride), petroleum hydrocarbons from cleaning solvents,
- Oil and grease, PAHs, and PCBs from oil wastes and cleaning rags,
- Beryllium in wastewater from cleaning aircraft parts (e.g., brakes),
- Asbestos from aircraft installation, and
- Mercury from contaminated rags or equipment.

2.4.9 Building 360

Available data indicate the presence of cyanide and 1,1,1-trichloroethane in at least one subsurface soil sample. Additional subsurface soil samples are needed to characterize this study area as well as groundwater and surface soil samples. Additional chemical parameters, for example, metals, should be analyzed in all samples and are detailed below.

- Phenol, and other organic solvents (e.g., 1,1,1-trichloroethane, and carbon tetrachloride) in wastewaters from engine cleaning, paint stripping and painting operations,

- Petroleum hydrocarbons, PAHs, and PCBs in waste oil from the engine rework shop,
- Acids and bases from electroplating and engine cleaning operations, and
- Metals (e.g., cadmium, chromium, copper, tin, lead, and zinc) and cyanide from electroplating operations.

2.4.10 Building 410

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams and are chemicals of potential concern:

- Phenol, and other organic solvents (e.g., methylene chloride), and metals (e.g., chromium) in wastewaters from paint stripping operations and
- Petroleum hydrocarbons, metals, PAHS, and PCBs from oil wastes.

2.4.11 Buildings 400 and 530 (Missile Rework Operations)

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams and are chemicals of potential concern:

- Phenol, and other organic solvents (e.g., methylene chloride, 1,1,1-trichloroethane, carbon tetrachloride), and metals in wastewater from paint stripping and painting operations and
- Petroleum hydrocarbons, metals, PAHs, PCBs from waste oil.

2.4.12 Building 14 (Test Shop)

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams and are chemicals of potential concern:

- Metals (e.g., mercury).

2.4.13 Buildings 301 and 389

Shallow soil samples indicated the presence of PCBs (Table 2-7) in this study area. However, di- and trichlorobenzenes, a common solvent for Arochlor 1260 in transformer fluids, were not included in the analytical parameters for this initial screening. Metals could also be potential contaminants in used transformers. Subsurface soils and groundwater samples should be analyzed for the above chemicals in the RI. At least three grab background samples should be collected in each medium and analyzed for the same chemical parameters as the samples.

2.4.14 Cans C-2 Area

Ten shallow surface and one subsurface soil samples and one groundwater sample contained metals, at least one pesticide, and a trace of gasoline hydrocarbons (Table 2-7). Base, neutral and acid extractables, volatile organic compounds and oil and grease should be included in the analytical parameters in the RI. Additional samples, particularly subsurface soils and groundwater, are needed to characterize this study area. At least three background samples should be collected in each medium and analyzed for the same chemical parameters as the samples.

Based on the past activities, the following chemicals have been identified as potential chemicals of concern in this area.

- Metals (e.g., barium, vanadium, cadmium, chromium, cobalt, copper, lead, mercury, nickel, vanadium, and zinc) from electroplating and paint stripping baths,
- Petroleum hydrocarbons, metals, PCBs, chlorobenzenes, and PAHs from miscellaneous oil wastes and leakage from disused equipment (e.g., transformers), and
- Organic solvents (e.g., 1,2-dichloroethylene) and pesticides (e.g., endrin, lindane, and 2,4-D) from stored wastes (e.g., paint stripper chemicals, pesticide containers).

2.4.15 Station Sewer System

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams and are chemicals of potential concern:

- Petroleum hydrocarbons, metals, PAHs, and PCBs from oily wastes,
- Phenols and other organic solvents and metals in wastewater from paint stripping operations,
- Cyanide, acids, bases, and metals (e.g., chromium) in wastewater from metal plating operations, and
- Pesticides from rinsing pesticide applicator equipment.

2.4.16 Seaplane Lagoon

Eight shallow sediment samples contained measurable amounts of metals, but organochlorine pesticides and PCBs were not measured above the quantification level (Table 2.7 and Section 2.3.16). Volatile organic compounds, base, neutral and acid extractable compounds, and oil and grease should also be included in RI sampling based on chemicals identified in past waste streams which emptied into the lagoon. Surface water samples at different depths, and additional sediment samples at different depths, are needed to characterize this study area. At least three background samples from each medium at each depth should be analyzed for the same chemical parameters as the samples.

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams which emptied into the Seaplane Lagoon:

- Metals (e.g., arsenic, barium, selenium, thallium, cadmium, chromium, cobalt, copper, lead, mercury, nickel, and zinc) in wastewaters from paint stripping and electroplating operations,.
- Petroleum hydrocarbons, metals, PAHs, and PCBs from oily wastes,

- Phenols and other organic solvents and metals in wastewater from paint stripping operations,
- Cyanide, acids, and bases in wastewater from metal plating operations,
- Pesticides from rinsing pesticide applicator equipment, and
- Tributyltin from the paint on the bottom of boats reported to have dissolved in the past (Alliance/WESTDIV, 1987).

2.4.17 Estuary (Oakland Inner Harbor)

Metals and petroleum hydrocarbons were detected in 8 sediment cores collected in the Oakland Inner Harbor as part of another study (U.S. Corp of Engineers 1988; see Table 2-7). Surface water and additional sediment samples and background samples should be collected at different depths as noted for the Seaplane Lagoon.

As discussed in Section 2.1, the following chemicals have been stored or identified in the waste streams which emptied into the Estuary:

- Metals (e.g., arsenic, cadmium, chromium, copper, lead, nickel, silver, and zinc) in wastewaters from paint stripping and electroplating operations,
- Petroleum hydrocarbons, metals, PAHs, and PCBs from oily wastes,
- Phenols and other organic solvents in wastewater from paint stripping operations,
- Cyanide, acids, and bases in wastewater from metal plating operations, and
- Pesticides from rinsing pesticide applicator equipment.

2.4.18 1943-1956 Disposal Area

Five shallow groundwater and five subsurface soil samples contained measurable amounts of metals, volatile organic compounds, base, acid/neutral extractable compounds, and radiation. Additional surface soil, subsurface

soil, and groundwater samples are necessary to adequately characterize this approximately 120-acre study area. At least 3 background samples from each medium at each depth should be analyzed for the same chemical parameters as the samples.

As discussed in Section 2.1, the following chemicals have been buried in the landfill (chemicals detected in subsurface soil or groundwater samples are listed in parentheses):

- Metals (e.g., arsenic, barium, cadmium, chromium, cobalt, copper, lead, mercury, molybdenum, nickel, and vanadium, zinc) from paint stripping and electroplating wastes,
- Organic solvents (e.g., acetone, 1,1,1-trichloroethane, 1,2-dichloroethylene, and benzene) from paints and paint stripping wastes,
- PAHs, PCBs, phthalates and ketones (e.g., bis(2-ethylhexyl)phthalate, di-n-butyl phthalate, 2-cyclohexen-1-one and 2.5-diethyltetrahydrofuran, acenaphthene, naphthalene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(g,h,i)perylene, acenaphthylene, pyrene, and dibenzofuran, benzo(a)pyrene, indeno(1,2,3-c,d)pyrene, chrysene, fluorene, phenanthrene, dibenzofuran and 2-methylnaphthalene) from oil and solvent wastes,
- Pesticides from pesticide wastes,
- Gross alpha and gross beta radiations from radiological wastes, and
- Asbestos from insulation wastes.

2.4.19 West Beach Landfill

Detected chemicals from groundwater sampling efforts are summarized in Table 2-7. Surface soil and subsurface soil samples are needed to characterize this study area. Surface water and sediment samples from the wetlands area within this study are also recommended. At least 3 background

samples from each depth sampled should be analyzed for the same chemical parameters as the samples.

As discussed in Section 2.1, the following chemicals have been buried in the landfill (chemicals detected in shallow groundwater are listed in parentheses):

- Metals (e.g., antimony, arsenic, beryllium, magnesium, selenium, and thallium, cadmium, chromium, copper, lead, mercury, nickel, and zinc) from paint stripping and electroplating wastes,
- Organic solvents (e.g., benzene, chlorobenzene, ethylbenzene, toluene, acetone, o-xylene, phenol, 2,4-dimethylphenol) from paints and painting wastes,
- PAHs, PCBs, phthalates and ketones (e.g., 2-methylnaphthalene, naphthalene, and bis(2-ethylhexyl)phthalate) from oil, creosote, and solvent wastes,
- Pesticides (e.g., d-BHC, endrin aldehyde, endosulfan sulfate, a-BHC, Heptachlor, g-BHC (lindane), Aldrin) from pesticide wastes,
- Cyanide, acids and bases from electroplating wastes,
- Asbestos from insulation wastes, and
- Gross alpha and gross beta radiation from radiological wastes.

2.4.20 Yard D-13

As discussed in Section 2.1, the following chemicals have been stored at Yard D-13 and are chemicals of potential concern:

- Metals, cyanide, organic solvents, pesticides, PCBs, and PAHs from stored hazardous wastes.

3.0 TOXICITY CHARACTERIZATION

In this section, brief descriptions of the human toxicity of the chemicals of potential concern at NAS Alameda are presented together with available standards, criteria, and toxicity values which have been developed for evaluation of exposure to these chemicals under specific circumstances. Due to the large number of potential contaminants of concern at NAS Alameda, the toxicity descriptions have been categorized into smaller groups (organics, herbicides, chlorinated hydrocarbons, etc.).

3.1 HEALTH EFFECTS CLASSIFICATION AND CRITERIA DEVELOPMENT

For risk assessment purposes, individual pollutants are separated into two categories of chemical toxicity, depending on whether they exhibit noncarcinogenic or carcinogenic effects. This distinction relates to the currently held scientific opinion that the mechanism of action for each category is different. EPA has adopted, for the purpose of assessing risks associated with potential carcinogens, the scientific position that a small number of molecular events can cause changes in a single cell or a small number of cells that can lead to tumor formation. This is described as a no-threshold mechanism, since there is essentially no level of exposure (i.e., a threshold) to a carcinogen which will not result in some finite possibility of causing the disease. In the case of chemicals exhibiting noncarcinogenic effects, however, it is believed that organisms have protective mechanisms that must be overcome before the toxic endpoint is manifested. For example, if a large number of cells performs the same or similar functions, it would be necessary for significant damage or depletion of these cells to occur before an effect could be seen. This threshold view holds that a range of exposures from just above zero to some finite value can be tolerated by the organism without appreciable risk of causing the disease (EPA, 1987s).

3.1.1 Health Effects Criteria for Noncarcinogens

Health criteria for chemicals exhibiting noncarcinogenic effects are generally developed using reference doses (RfDs) developed by the EPA RfD Work Group, or RfDs obtained from EPA Health Effects Assessments (HEAs). The RfD, expressed in units of mg/kg/day, is an estimate of the daily exposure to the human population (including sensitive subpopulations) that is likely to be without an appreciable risk of deleterious effects during a lifetime. These RfDs are usually derived either from human studies involving workplace exposures or from animal studies, and are adjusted using uncertainty factors. The RfD provides a benchmark to which chemical intakes by other routes (e.g., via exposure to contaminated environmental media) may be compared.

3.1.2 Health Effects Criteria for Potential Carcinogens

Cancer potency factors, developed by EPA's Carcinogen Assessment Group (CAG) for potentially carcinogenic chemicals and expressed in units of $(mg/kg/day)^{-1}$, are derived from the results of human epidemiological studies or chronic animal bioassays. The animal studies must usually be conducted using relatively high doses in order to detect possible adverse effects. Since humans are expected to be exposed at lower doses than those used in the animal studies, the data are adjusted by using mathematical models. The data from animal studies are typically fitted to a linearized multistage model to obtain a dose-response curve. The 95th percentile, upper confidence limit slope of the dose-response curve is subjected to various adjustments and an interspecies scaling factor is usually applied to derive the cancer potency factor for humans. Thus, the actual risks associated with exposure to a potential carcinogen quantitatively evaluated based on animal data are not likely to exceed the risks estimated using these cancer potency factors, but they may be much lower. Dose-response data derived from human epidemiological studies are fitted to dose-time-response curves on an ad hoc basis. These models provide rough, but plausible, estimates of the upper limits on lifetime risk. Cancer potency factors based on human epidemiological data are also derived using very conservative assumptions and, as such, they too are

unlikely to underestimate risks. Therefore, while the actual risks associated with exposures to potential carcinogens are unlikely to be higher than the risks calculated using a cancer potency factor, they could be considerably lower.

EPA assigns weight-of-evidence classifications to potential carcinogens. Under this system, chemicals are classified as either Group A, Group B1, Group B2, Group C, Group D, or Group E. Group A chemicals (known human carcinogens) are agents for which there is sufficient evidence to support the causal association between exposure to the agents in humans and cancer. Groups B1 and B2 chemicals (probable human carcinogens) are agents for which there is limited (B1), or inadequate (B2) evidence of carcinogenicity from human studies, but for which there is sufficient evidence of carcinogenicity from animal studies. Group C chemicals (possible human carcinogens) are agents for which there is limited evidence of carcinogenicity in animals, and Group D chemicals (not classified as to human carcinogenicity) are agents with inadequate human and animal evidence of carcinogenicity or for which no data are available. Group E chemicals (evidence of non-carcinogenicity in humans) are agents for which there is no evidence of carcinogenicity in adequate human or animal studies.

Tables 3-1 and 3-2 present oral and inhalation toxicity values for the contaminants of concern at NAS Alameda.

3.1.3 Regulatory and Occupational Safety Standards

Included in these chemical descriptions are State regulatory and Federal safety standards. The California State Applied Action Levels (AALs) were developed by the Department of Health Services for the protection of human health from chemicals in environmental media (CDHS, 1989). These have been included for the chemicals of potential concern in water and air when available.

Table 3-1
Critical Oral Toxicity Values
Alameda Naval Air Station

Chemical	RfD (mg/kg/day)	[Uncertainty Factor][a]	Source[b]	Cancer Potency Factor $(mg/kg/day)^{-1}$	EPA Weight of Evidence[c]
ORGANICS					
Acetone	1×10^{-1}	[1,000]	IRIS	—	—
Carbon tetrachloride	7×10^{-4}	[1,000]	IRIS	1.3×10^{-1}	B2
1,2-Dichloroethylene	1×10^{-2}	[1,000]	HEA	—	—
2,4-Dimethylphenol	—		—	—	—
Methylene chloride	6×10^{-2}	[100]	IRIS	7.5×10^{-3}	B2
Methyl ethyl ketone	5×10^{-2}	[1,000]	IRIS	—	—
Phenol	4×10^{-2}	[1,000]	IRIS	—	—
Tetrachloroethylene	1.0×10^{-2}	[1,000]	IRIS	5.1×10^{-2}	B2
1,1,1-Trichloroethane	9.0×10^{-2}	[1,000]	IRIS	—	—
Trichloroethylene	7.4×10^{-3}		HEA	1.1×10^{-2}	B2
Vinyl chloride	—		—	2.3×10^{0}	A
BTEX COMPOUNDS					
Benzene	—		—	2.9×10^{-2}	A
Chlorobenzene	2.7×10^{-2}		HEA	—	—
Ethylbenzene	1.0×10^{-1}	[1,000]	IRIS	—	—
Toluene	3×10^{-1}	[100]	IRIS	—	—
Xylene	2×10^{0}	[100]	IRIS	—	—
PESTICIDES					
Aldrin	3×10^{-5}		IRIS	1.7×10^{1}	B2
Bromacil	—		—	—	—
Chlordane	5×10^{-5}	[1,000]	IRIS	1.3×10^{0}	B2
Chlorvar	—		—	—	—
Diazinon	9×10^{-5}	[100]	HEA	—	—
2,4-D	1×10^{-2}	[100]	IRIS	—	—
DDT	5×10^{-4}	[100]	IRIS	3.4×10^{-1}	B2
Diuron	2×10^{-3}	[300]	IRIS	—	—
Endosulfan	5×10^{-5}		IRIS	—	—

Table 3-1 -Continued-
Critical Oral Toxicity Values
Alameda Naval Air Station

Chemical	RfD (mg/kg/day) [Uncertainty Factor][a]	Source[b]	Cancer Potency Factor $(mg/kg/day)^{-1}$	EPA Weight of Evidence[c]
PESTICIDES (cont.)				
Endrin	3×10^{-4} [100]	IRIS	—	—
Heptachlor	5×10^{-4} [300]	IRIS	4.5×10^{0}	B2
Krovar I	—	—	—	—
Lindane	3×10^{-4} [1,000]	HEA	1.3×10^{0}	B2-C
Malathion	2×10^{-2} [10]	IRIS	—	—
Monuron	—	—	—	—
Roundup	1×10^{-1} [100]	IRIS	—	—
Simazine	5×10^{-3} [1,000]	HEA	—	—
Telvar	—	—	—	—
Warfarin	3×10^{-4}	IRIS	—	—
INORGANICS				
Antimony	4×10^{-4} [1,000]	IRIS	—	—
Arsenic	—	—	1.75×10^{0}	A
Barium	5×10^{-2} [100]	IRIS	—	—
Beryllium	5×10^{-3} [100]	IRIS	—	—
Cadmium	5×10^{-4}* [10]	HEA	—	—
Chromium III	1×10^{0} [100]	IRIS	—	—
Chromium VI	5×10^{-3} [500]	IRIS	—	—
Cobalt	—	—	—	—
Copper	3.7×10^{-2} [2]	HEA	—	—
Iron	—	—	—	—
Lead	—	—	**	B2
Manganese	2.1×10^{-1} [100]	HEA	—	—
Mercury ,Inorganic	2×10^{-3} [1,000]	HEA	—	—
Mercury, Organic	3×10^{-4} [10]	HEA	—	—
Nickel	2×10^{-2} [100]	IRIS	—	—
Selenium	3×10^{-3} [15]	HEA	—	—

Table 3-1 -Continued-
Critical Oral Toxicity Values
Alameda Naval Air Station

Chemical	RfD (mg/kg/day) [Uncertainty Factor][a]		Source[b]	Cancer Potency Factor $(mg/kg/day)^{-1}$	EPA Weight of Evidence[c]
INORGANICS (cont.)					
Silver	3×10^{-3}	[2]	IRIS	—	—
Thallium	7×10^{-4}	[1,000]	HEA	—	—
Tin	6×10^{-1}	[100]	IRIS	—	—
Vanadium	5.7×10^{-3}		HEA	—	—
Zinc	2×10^{-1}	[10]	HEA	—	—
OTHERS					
Asbestos	—		—	—‡	A
Bis (2-ethylhexyl)phthalate	2 x 10-2	[1,000]	IRIS	8.4×10^{-3}	B2
Cyanides	2×10^{-2}	[500]	IRIS	—	—
PCBs	—		—	7.7×10^{0}	B2
PAHs - Noncarcinogenic	4.1×10^{-1}	[100]	HEA	—	—
- Carcinogenic	—		—	1.15×10^{1}	B2

a) Uncertainty factors used to develop reference doses consist of multiples of 10, each factor representing a specific area of uncertainty inherent in the data available. The standard uncertainty factors include:

- a ten-fold factor to account for the variation in sensitivity among the members of the human population;
- a ten-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;
- a ten-fold factor to account for uncertainty in extrapolating from less than chronic NOELs to chronic NOAELs; and
- a ten-fold factor to account for uncertainty in extrapolating from LOAELS to NOAELs.

b) Sources of reference doses: IRIS = chemical files of the Integrated Risk Information System; HEA = Health Effects Assessment.

c) Weight of evidence classification schemes for carcinogens: A — Human Carcinogen, sufficient evidence from human epidemiological studies; B1 -- Probable Human Carcinogen, limited evidence from epidemiological studies

Table 3-1 -Continued-
Critical Oral Toxicity Values
Alameda Naval Air Station

and adequate evidence from animal studies; B2 -- Probable Human Carcinogen, inadequate evidence from epidemiological studies and adequate evidence from animal studies; C -- Possible Human Carcinogen, limited evidence in animals in the absence of human data.

*This RfD is for drinking water exposures. An RfD of 1 x 10^{-3} mg/kg/day has been derived for other nonaqueous exposure sources (EPA 1988b).
**Certain Lead salts have been determined to be carcinogenic, but EPA has not derived cancer potency factors for these compounds.

‡A unit risk cancer potency factor of 1.4 x 10^{-13} (fiber/liter)$^{-1}$ has been derived for asbestos.

Table 3-2
Inhalation Toxicity Values
Alameda Naval Air Station

Chemical	Reference Dose (mg/kg/day) [Uncertainty Factor][a]	Source[b]	Cancer Potency Factor $(mg/kg/day)^{-1}$	EPA Weight of Evidence
ORGANICS				
Acetone	--	--	--	--
Carbon Tetrachloride	--	--	1.3×10^{-1}	B2
1,2-Dichloroethylene	--	--	1.2×10^{0}	C
2,4-Dimethylphenol	--	--	--	--
Methyl ethyl ketone	9×10^{-2} [1,000]	HEA	--	--
Methylene chloride	--	--	1.4×10^{-2}	B2
Phenol	2×10^{-2}	HEA	--	--
Tetrachloroethylene	--	--	3.3×10^{-3}	B2
1,1,1-Trichloroethane	3×10^{-1} [1,000]	HEA	--	--
Trichloroethylene	--	--	4.6×10^{-3}	B2
Vinyl Chloride	--	--	2.9×10^{-1}	A
BTEX COMPOUNDS				
Benzene	--	--	2.9×10^{-2}	A
Chlorobenzene	5.7×10^{-3}	HEA	--	--
Ethylbenzene	--	--	--	--
Toluene	1.0×10^{0} [100]	HEA	--	--
Xylene	4.0×10^{-1} [1,000]	HEA	--	--
PESTICIDES				
Aldrin	--	--	1.7×10^{1}	B2
Bromacil	--	--	--	--
Chlordane	--	--	1.3×10^{0}	B2
Chlorvar	--	--	--	--
Diazinon	--	--	--	--
2,4-D	--	--	--	--
DDT	--	--	--	--
Diuron	--	--	--	--
Endosulfan	--	--	--	--
Endrin	--	--	--	--
Heptachlor	--	--	4.5×10^{0}	B2
Krovar I	--	--	--	--
Lindane	--	--	--	--
Malathion	--	--	--	--
Monuron	--	--	--	--
Roundup	--	--	--	--
Simazine	--	--	--	--
Telvar	--	--	--	--
Warfarin	--	--	--	--

Table 3-2 -Continued-
Inhalation Toxicity Values
Alameda Naval Air Station

Chemical	Reference Dose (mg/kg/day) [Uncertainty Factor][a]	Source[b]	Cancer Potency Factor $(mg/kg/day)^{-1}$	EPA Weight of Evidence
INORGANICS				
Antimony	--	--	--	--
Arsenic	--	--	5×10^{1}	A
Barium	1.4×10^{-4} [1,000]	HEA	--	--
Beryllium	--	--	8.4×10^{0}	B2
Cadmium	--	--	6.1×10^{0}	B1
Chromium III	--	--	--	--
Chromium VI	--	--	4.1×10^{1}	A
Cobalt	--	--	--	--
Copper	--	--	--	--
Iron	--	--	--	--
Lead	--	--	--	--
Manganese	3×10^{-4} [100]	HEA	--	--
Mercury, Inorganic	--	--	--	--
Mercury, Organic	--	--	--	--
Nickel	--	--	--[c]	--[c]
Selenium	1×10^{-3} [10]	HEA	--	--
Silver	--	--	--	--
Thallium	--	--	--	--
Tin	--	--	--	--
Vanadium	--	--	--	--
Zinc	--	--	--	--
OTHERS				
Asbestos	--	--	2.3×10^{-1}	A
Bis(2-ethylhexyl) phthalate	--	--	--	--
Cyanides	--	--	--	--
PCBs	--	--	--	--
PAHs - Noncarcinogenic	--	--	--	--
- Carcinogenic	--	--	6.1×10^{0}	B2

a) Uncertainty factors used to develop reference doses consist of multiples of 10, each factor representing a specific area of uncertainty inherent in the data available. The standard uncertainty factors include:
- a ten-fold factor to account for the variation in sensitivity among the members of the human population;
- a ten-fold factor to account for the uncertainty in extrapolating animal data to the case of humans;

Table 3-2 -Continued-
Inhalation Toxicity Values
Alameda Naval Air Station

Chemical	Reference Dose (mg/kg/day) [Uncertainty Factor][a]	Source[b]	Cancer Potency Factor $(mg/kg/day)^{-1}$	EPA Weight of Evidence

- a ten-fold factor to account for uncertainty in extrapolating from less than chronic NOELs to chronic NOAELs; and
- a ten-fold factor to account for uncertainty in extrapolating from LOAELS to NOAELs.

b) Sources of reference doses: IRIS = chemical files of the Integrated Risk Information System; HEA = Health Effects Assessment.

c) EPA has derived individual cancer potency factors for nickel refinery dust (0.84 $(mg/kg/day)^{-1}$) and nickel subsulfide (1.7 $(mg/kg/day)^{-1}$) and has classified these substances in Group A. EPA has not classified or derived a cancer potency factor for elemental nickel.

The Federal Occupational Safety and Health Administration (OSHA) revised the air contaminant standards (29 CFR 1910.1000) in March 1989 which establish acceptable airborne concentrations in the workplace to protect employee health (OSHA, 1989). The new standards will take effect on September 1, 1989 and are cited in this document. Average, short term, and ceiling levels of airborne chemicals have been defined for employee exposure. In addition, a "skin" designation has been established to protect workers against exposure by skin contact. When these levels have been available for the chemicals of potential concern, they have been included.

The 8-hour Time Weighted Average (TWA) permissible exposure limit (PEL) is the average chemical concentration which an employee can be exposed to over the course of a 8-hour work period over a 40-hour work week. This ensures protection from all ill health effects potentially caused by the chemical.

Short Term Exposure Limits (STELs) are 15-minute time weighted average exposure concentrations. These concentrations are not to be exceeded for more than 15-minutes each day in order for the workplace to remain protective of employee health.

Ceiling limits have also been developed for some chemicals. Ceiling limits are not to be exceeded at any time during any part of the working day.

In some instances, a "skin" designation has been assigned to certain chemicals. This designation is assigned if a chemical is known to cause ill effects when exposed to skin, eyes, or mucous membranes. Use of protective clothing, gloves, and goggles are recommended for employees involved in work including these chemicals. Chemicals of potential concern with this designation have been mentioned as such in each description.

ORGANIC COMPOUNDS

Monocyclic Aromatic Hydrocarbons

BENZENE

Benzene is readily absorbed following oral and inhalation exposure (EPA, 1985a). The toxic effects of benzene in humans and other animals following exposure by inhalation include central nervous system effects, hematological effects, and immune system depression (ATSDR, 1987c). In humans, acute exposure to high concentrations of benzene vapors has been associated with dizziness, nausea, vomiting, headache, drowsiness, narcosis, coma, and death (NAS, 1976). Inhalation of benzene can cause eye and respiratory system irritation (NIOSH, 1985b). Chronic exposure to benzene vapors can produce reduced leukocyte, platelet, and red blood cell counts (EPA, 1985a). Inhalation experiments conducted in rats, mice, guinea pigs, and rabbits suggest that benzene is not teratogenic at doses that are fetotoxic and embryolethal (IARC, 1982). It has been shown to be embryo/fetotoxic at maternally toxic dose levels and it is a potent inhibitor of growth and development in utero (EPA, 1985a). Increased incidences of fetal resorptions, skeletal variations, and altered fetal hematopoiesis have been reported (ATSDR, 1987c). Animal experiments in rats, guinea pigs, and rabbits suggest that exposures to benzene vapors may damage the testes of adult males (IARC, 1982).

Epidemiological studies in occupational settings have described a causal relationship between exposure to benzene by inhalation (either alone or in combination with other chemicals) and leukemia in humans (IARC, 1982). Benzene has also been shown to induce both solid tumors and leukemias and lymphomas in rats and mice exposed by gavage (Maltoni et al., 1985; NTP, 1986) and leukemias and lymphomas in mice exposed by inhalation (Snyder et al., 1980, Cronkite et al., 1985). EPA (1986a) has classified benzene in Group A-Human Carcinogen based on adequate evidence of carcinogenicity from epidemiological studies. EPA (1988a) derived both an oral and an inhalation

cancer potency factor for benzene of 2.9×10^{-2} $(mg/kg/day)^{-1}$. This value was based on several studies in which increased incidences of nonlymphocytic leukemia were observed in humans occupationally exposed to benzene principally by inhalation (Rinsky, 1981; Ott, 1978; Wong, 1983). A time weighted average concentration of 10 ppm has been determined for benzene as well as a ceiling limit of 25 ppm (OSHA, 1989). Applied action levels of 0.7 $\mu g/L$ of water and 3.2 $\mu g/m^3$ of air have been established for benzene under the California Department of Health Services (CDHS, 1988c).

CHLOROBENZENE

Evidence from toxicity studies suggests that chlorobenzene is absorbed after oral, inhalation, and dermal exposure (EPA, 1985w). Symptoms following inhalation and ingestion exposure include irritation of eyes, nose, and skin, drowsiness, and incoherence. Acute and chronic exposure to chlorobenzene has been associated with central nervous system effects, liver and kidney lesions, and respiratory distress in humans and experimental animals. Results of reproductive studies with rats and dogs also indicate that chlorobenzene induces testicular lesions (EPA, 1985w).

Chlorobenzene has been tested in rodent carcinogenesis bioassays. Negative results were reported in both sexes of mice and in male rats and an equivocal increase in liver tumors was seen in female rats. EPA (1985w) considered these results to be insufficient to conclude whether or not chlorobenzene is carcinogenic.

EPA (1984w) derived an oral RfD for exposure to chlorobenzene of 2.7×10^{-2} mg/kg/day based on a study by Monsanto (1967). EPA (1984w) also derived an inhalation AIC for chlorobenzene of 5.7×10^{-3} mg/kg/day based on a study by Dilley (1977). The time weighted average by OSHA has been set at 75 ppm (350 mg/m^3) for the protection of employee health (OSHA, 1989).

ETHYLBENZENE

Ethylbenzene is primarily absorbed via inhalation and distributed throughout the body in rats; the highest levels were detected in the kidney, lung, adipose tissue, digestive tract, and liver (Chin et al., 1980). In humans, short-term inhalation and ingestion exposure can result in drowsiness, fatigue, coma, headache, dermatitis, and mild eye and respiratory irritation (Bardodej and Bardodejova, 1970; NIOSH, 1985b). Human exposure to high concentrations of ethylbenzene may cause central nervous system effects (NIOSH, 1985). Eye irritation has also been observed in experimental animals exposed to ethylbenzene (EPA, 1987k). Acute exposure to rats results in systemic effects primarily of the liver and kidney (Wolf et al., 1956). Chronic oral exposure of rats also results in adverse hepatic and renal effects including increased organ weights and cloudy swelling (Wolf et al., 1956). Ethylbenzene was not embryotoxic, teratogenic, or maternally toxic for New Zealand white rabbits; maternal toxicity was observed in rats (Hardin et al., 1981). No information on the carcinogenic potential of ethylbenzene was located in the reviewed literature.

EPA (1988a) derived an oral RfD of 0.1 mg/kg/day for ethylbenzene based on a study in which no liver or kidney toxicity was observed in rats exposed to 136 mg/kg/day (Wolf et al., 1956). An uncertainty factor of 1,000 was used to derive the reference dose.

A final ruling by OSHA has set the 8-hour TWA at 100 ppm (435 mg/m^3), and the STEL at 125 ppm (545 mg/m^3) for ethylbenzene (OSHA, 1989). An applied action limit for water of 0.68 mg/L and an applied action level for air of 0.14 mg/m^3 has been set by DHS for ethylbenzene (DHS, 1988c).

TOLUENE

Toluene is absorbed in humans following both inhalation and dermal exposure (EPA, 1985b). In humans, the primary acute effects of exposure to toluene vapor are central nervous system (CNS) depression and narcosis. These

effects occur at concentrations of $\geq$ 200 ppm (754 mg/m^3) (von Oettingen et al., 1942a,b). Symptoms of acute exposure to toluene in humans include fatigue, weakness, dizziness, headache, confusion, and euphoria for inhalation exposure; dilated pupils and lacrimation (tearing) for dermal absorption; and nervousness, muscle fatigue, and insomnia for ingestion (NIOSH, 1985b). In experimental animals, acute oral and inhalation exposures to toluene can result in CNS depression and lesions of the lungs, liver, and kidneys (EPA, 1987h). The earliest observable sign of acute oral toxicity in animals is inhibition of the CNS, which becomes evident at approximately 2,000 mg/kg (Kimura et al., 1971). In humans, chronic exposure to toluene vapors at concentrations of approximately 200 and 800 ppm has been associated with CNS and peripheral nervous system effects, hepatomegaly, and hepatic and renal function changes (EPA, 1987h). Toxic effects following prolonged exposure of experimental animals to toluene are similar to those seen following acute exposure (Hanninen et al., 1976; von Oettingen et al., 1942a). There is some evidence that oral exposure to greater than 0.3 ml/kg toluene during gestation results in embryotoxicity in CD-1 mice (Nawrot and Staples, 1979). Inhalation exposure of up to 1,000 mg/m^3 by pregnant rats during gestation has been associated with significant increases in skeletal retardation (Hudak and Ungvary, 1978).

Toluene has not been shown to be carcinogenic. No tumors were induced in rats exposed to toluene vapors for up to 24 months (CIIT, 1980).

EPA (1988a) derived an oral RfD for toluene based on a 24-month inhalation study in which rats were exposed to concentrations as high as 300 ppm (30 mg/kg/day) (CIIT, 1980). No adverse effects were observed in any of the treated animals. Using the no-observed-effect level (NOEL) of 30 mg/kg/day and an uncertainty factor of 100, an oral RfD of 0.3 mg/kg/day was derived. EPA (1988b) reported an inhalation RfD for toluene of 1 mg/kg/day also based on this CIIT study and using an uncertainty factor of 100. Toluene has a time weighted average of 100 ppm (375 mg/m^3) set by OSHA. The STEL for toluene has been set at 150 ppm (560 mg/m^3) (OSHA, 1989). No ceiling exists for this chemical. Applied action levels of 0.10 mg/L and 0.20

mg/m^3 have been set for water and air concentrations of toluene, respectively (DHS, 1988c).

XYLENES

Metabolism and excretion studies suggest that orally administered xylene is nearly completely absorbed. Acute exposure to relatively high concentrations of xylene causes central nervous system depression, minor reversible effects on the liver and kidneys, and irritation of the eyes, nose, and throat. Target organs of xylene include the gastrointestinal tract, blood, liver, and kidneys (NIOSH, 1985). The most common symptoms found in people occupationally exposed to xylene are headache, fatigue, lassitude, irritability, and gastrointestinal disorders, including nausea, anorexia and flatulence (ACGIH, 1986). Other symptoms of acute exposure include incoherence, staggering gait and excitement (NIOSH, 1985b). The liver is reportedly affected by longer-term exposure to lower levels of xylene (EPA, 1984x, 1985a).

The National Toxicology Program (NTP) reported that oral administration of mixed xylenes does not result in tumor formation in rats or mice (NTP, 1986c).

EPA (1984x) derived an inhalation RfD of 4×10^{-1} mg/kg/day based on an inhalation study by Jenkins et al. (1970). An oral RfD of 2 mg/kg/day was derived by EPA (1987m) based on the National Toxicology Program carcinogenesis bioassay (1986c). Worker exposure limits for xylene have been set by OSHA at 100 ppm (435 mg/m^3) for the 8-hour time weighted average and 150 ppm (655 mg/m^3) for the STEL (OSHA, 1989). No ceiling limit has been set for xylene. Water concentrations of 0.62 mg/l and ambient air concentrations of 0.10 mg/m^3 have been developed as applied action levels by the DHS (1988c).

Phenolics

2,4-DIMETHYLPHENOL

2,4-Dimethylphenol (2,4-DMP) is absorbed primarily through the skin by humans occupationally exposed to mixtures containing 2,4-DMP. Clinical signs of acute poisoning by methylphenols in mice, rats, and rabbits include dyspnea, disturbance of motor coordination, and rapid onset of clonic spasms (Maazik, 1968). Signs observed during inhalation of 2,4-DMP include irritation of mucous membranes, enlargement of blood vessels in ears and extremities, and excitation followed by lethargy (Uzhdovini et al., 1974). Male rats treated with two different dimethylphenol isomers (3,4- and 2,6-isomers) for 10 weeks in the diet exhibited depressed weight gains and increased organ-to-body weight ratios for the liver, spleen, heart, and lungs. Atrophy and dystrophy of hepatic cells were also noted. No information on the teratogenic or genotoxic potential of 2,4-DMP was found in the literature reviewed (EPA, 1980h). Boutwell and Bosch (1959) reported that papillomas and carcinomas were produced on the skin of mice dermally exposed to high concentrations of 2,4-DMP. EPA has not promulgated any RfDs or cancer potency factors for 2,4-DMP. No worker exposure limits have been set by OSHA for 2,4-dimethylphenol.

PHENOL

Phenol is readily absorbed through the gastrointestinal tract, by inhalation, and percutaneously (EPA, 1980a). Signs of acute phenol toxicity in humans and experimental animals are central nervous system depression, collapse, coma, cardiac arrest, and death. Other symptoms include irritation of eye, nose and throat due to inhalation, muscle ache and pain due to ingestion, and dermatitis due to direct contact (NIOSH, 1985b). Acutely toxic doses can also cause extensive necrosis at the site of exposure (eyes, skin, oropharynx) (EPA, 1980j). In experimental animals, subchronic oral and inhalation studies suggest that kidney, pulmonary, myocardial, and liver damage are associated with exposure, although many of these studies were

poorly designed (EPA, 1980j, 1984t). Phenol has not been shown to be carcinogenic in rats or mice, but it has been observed to promote skin tumor development following initiation by other carcinogens. Phenol exhibited tumor-promoting activity in the mouse skin painting system following initiation with 9,10-dimethyl-1,2-benzanthracene (DMBA) or benzo[a]pyrene (B[a]P), and it exhibited cutaneous carcinogenic activity in a sensitive mouse strain when applied at concentrations that produced repeated skin damage (EPA, 1980a).

EPA (1988a) derived an oral RfD of 4.0×10^{-2} mg/kg/day based on an unpublished study reported by the Dow Chemical Company (1976) in which rats exposed to the lowest dose tested (50 mg/kg) exhibited renal and hepatic lesions. An inhalation RfD of 1.4 mg/person/day (0.02 mg/kg/day) was recommended by EPA (1984t) based on the threshold limit value of 19 mg/m^3 for phenol established by the American Conference of Governmental Industrial Hygienists (ACGIH, 1983). A time weighted average of 5 ppm (19 mg/m^3) has been promulgated by OSHA as a final rule limit on acceptable employee exposure levels for an 8-hour work day (OSHA, 1989).

Chlorinated Aliphatic Hydrocarbons

CARBON TETRACHLORIDE

Carbon tetrachloride (CCl_4) is readily absorbed following oral and inhalation exposure (EPA, 1984a). Carbon tetrachloride, like many other chlorinated hydrocarbons, acts as a central nervous system depressant (ACGIH, 1986). Symptoms of acute exposure to carbon tetrachloride include nausea, vomiting and skin irritation (NIOSH, 1985b). The toxic effects of oral and inhalation exposure to carbon tetrachloride in humans and animals include damage to the liver, kidney, and lung. The liver is the most sensitive tissue (EPA 1985c). In animals, acute oral administration of 100-4000 mg/kg/day produces fatty infiltration and histological alterations in the liver. High doses produce irreversible liver damage and necrosis while the effects observed following lower doses are largely reversible (EPA, 1985c). Humans

occupationally exposed to 5-15 ppm of carbon tetrachloride experienced less severe effects including biochemical alterations, nausea, headaches and in more severe cases, liver dysfunction (jaundice, enlargement, and fatty infiltration) (ACGIH, 1986; EPA, 1984a). Animals chronically exposed to carbon tetrachloride exhibit effects similar to those observed from acute exposures.

Prenatal toxicity has been demonstrated in mammalian fetuses and neonates after inhalation exposure of pregnant rats (EPA, 1984a), although carbon tetrachloride has not been shown to be teratogenic (EPA, 1985c).

Carbon tetrachloride is a carcinogen in animals producing mainly hepatic neoplasms. Doses of about 30 mg/kg/day or higher for 6 months have been found to produce an increased frequency of hepatomas, hepatocellular adenomas and hepatocellular carcinomas in mice, rats and hamsters (EPA, 1985c). EPA (1988a) has classified carbon tetrachloride as a Group B2 carcinogen - Probable Human Carcinogen, based on its carcinogenicity in experimental animals. EPA (1988a) established a cancer potency factor of 1.3×10^{-1} $(mg/kg/day)^{-1}$ for inhalation and oral exposure based on several studies in which hepatocellular carcinomas and hepatomas were observed in rats, mice and hamsters (Della Porta et al., 1961; Edwards et al., 1942; NCI, 1976a, 1977e).

EPA (1988a) has derived an oral RfD of 7×10^{-4} mg/kg/day based on a subchronic rat gavage study in which liver lesions were the most sensitive and critical effect (Bruckner et al., 1986). A no-observed-adverse-effect level (NOAEL) of 1.0 mg/kg/day was identified in this study and an uncertainty factor of 1000 was used to derive the RfD. OSHA has recently set a time weighted average of 2 ppm (12.6 mg/m^3) for the final rule limits of carbon tetrachloride (OSHA, 1989).

1,2-DICHLOROETHYLENE (cis- and trans- isomers)

cis-1,2-Dichloroethylene and trans-1,2-dichloroethylene are isomeric, synthetic chemicals. Because the toxicological information regarding the two isomers is similar, it is described here in the same section.

Specific data concerning the toxicokinetic activity cis- and trans-1,2-dichloroethylene are limited. Both cis- and trans-1,2-dichloroethylene should be rapidly absorbed by any route and the highest concentrations would be expected to be found in the liver and kidneys based on the known activity of similar chlorinated compounds (McKenna et al., 1978). Excretion of liver metabolites should be relatively rapid and predominantly through the urine (Jaegar et al., 1977). In humans, both isomers are central nervous system depressants at high concentrations (Irish, 1963). The trans- isomer has anaesthetic effects on humans at high doses (Irish, 1963). cis-1,2-Dichloroethylene has anaesthetic properties and has been found to cause nausea, vomiting, weakness, and cramps in humans (EPA, 1984b). 1,2-Dichloroethylene causes eye and respiratory system irritation following inhalation exposure in workers (NIOSH, 1985b). cis-1,2-Dichloroethylene also has narcotic effects in animals at high doses; acute exposure can cause narcosis and death in rats (Torkelson and Rowe, 1981).

No information on the teratogenic or carcinogenic potential of cis- or trans-1,2-dichloroethylene was found in the literature reviewed (EPA, 1987c,p).

EPA derived an oral RfD of 0.01 mg/kg/day for 1,2-dichloroethylene (both isomers) based on a 2-year chronic toxicity/oncogenicity assay (Quast et al., 1983) in which rats received 0, 50, 100, or 200 mg/liter 1,1-dichloroethylene in drinking water. This RfD was derived on the basis of 1,1-dichloroethylene because of the lack of toxicity information specific to 1,2-dichloroethylene; it was assumed that the effects of the two compounds should be similar. EPA (1988a) has derived an inhalation RfD of 1.2×10^{0} mg/kg/day for

1,2-dichloroethylene. 1,2-Dichloroethlylene has received a time weighted average under the final rule limits set by OSHA of 200 ppm (790 mg/m^3) (OSHA, 1989).

METHYLENE CHLORIDE

The absorption of ingested methylene chloride is virtually complete. The amount of airborne methylene chloride absorbed increases in direct proportion to its concentration in inspired air, the duration of exposure, and physical activity. Dermal absorption has not been accurately measured (EPA, 1985o). Because of methylene chloride's high solubility in water and lipids, it is probably distributed throughout all body fluids and tissues. Acute human exposure to methylene chloride may result in irritation of eyes, skin, and respiratory track; central nervous system depression; elevated carboxyhemoglobin levels; and circulatory disorders that may be fatal (EPA, 1985o). Ingestion of methylene chloride may cause numb or tingling limbs, and nausea, while inhalation may cause weakness, fatigue and sleepiness (NIOSH, 1985b). Chronic exposure of animals can produce renal and hepatic toxicity (EPA, 1985o).

EPA (1988a) classified methylene chloride in Group B2 – Probable Human Carcinogen. EPA (1985p) concluded that the induction of distant site tumors from inhalation exposure and the borderline significance for induction of tumors in a drinking water study are an adequate basis for concluding that methylene chloride be considered a probable human carcinogen via ingestion as well as inhalation. EPA (1985p) derived an inhalation cancer potency factor of 1.4×10^{-2} $(mg/kg/day)^{-1}$ based on the results of a National Toxicology Program (NTP) bioassay in which rats and mice were exposed to methylene chloride by inhalation for 6 hours a day, 5 days/week for 102 weeks. Significant increases in the incidence of mammary tumors in male and female rats and lung and liver tumors in male and female mice were reported. EPA (1985p) derived an oral cancer potency factor of 7.5×10^{-3} $(mg/kg/day)^{-1}$ based on the results of the NTP (1986a) inhalation bioassay and on an ingestion bioassay conducted by the National Coffee Association (NCA, 1983). In the NCA

study, groups of from 50 to 200 mice received methylene chloride in drinking water and a significant increase in the incidence of hepatocellular adenomas and/or carcinomas was reported for male mice.

An oral RfD of 0.06 mg/kg/day has been developed by EPA (1988a) based on a 2-year rat drinking water bioassay (NCA, 1982) that identified NOELS of 5.85 and 6.47 mg/kg/day for male and female rats, respectively. Liver toxicity was observed at doses of 52.58 and 58.32 mg/kg/day for males and females, respectively. Permissible exposure limit setting for methylene chloride by OSHA is currently underway and final rule PELS have not yet been set (OSHA, 1989).

TETRACHLOROETHYLENE

Tetrachloroethylene is absorbed following inhalation (IARC, 1979) and oral (EPA, 1985d,e) exposure. Tetrachloroethylene vapors and liquid also can be absorbed through the skin (EPA, 1985d,e). The principal toxic effects of tetrachloroethylene in humans and animals following acute and longer-term exposures include central nervous system depression and fatty infiltration of the liver and kidney with concomitant changes in serum enzyme activity levels indicative of tissue damage (EPA, 1985d,e). Symptoms of inhalation exposure by workers include irritation of eyes, nose and throat; ingestion causes nausea and flushness of the face and neck (NIOSH, 1985b). Humans exposed to doses of between 135 and 1,018 mg/m^3 for 5 weeks develop central nervous system effects, such as lassitude and signs of inebriation (Stewart et al., 1974). The offspring of female rats and mice exposed to high concentrations of tetrachloroethylene for 7 hours daily on days 6-15 of gestation developed toxic effects, including a decrease in fetal body weight in mice and a small but significant increase in fetal resorption in rats (Schwetz et al., 1975). Mice also exhibited developmental effects, including subcutaneous edema and delayed ossification of skull bones and sternebrae (Schwetz et al., 1975).

In a National Cancer Institute bioassay (NCI, 1977a), a high incidence of hepatocellular carcinoma was observed in both sexes of mice administered

tetrachloroethylene in corn oil by gavage 5 days per week for 78 weeks. Increased incidences of mononuclear cell leukemia and renal adenomas and carcinomas (combined) have also been observed in long term bioassays in which rats were exposed to tetrachloroethylene by inhalation (NTP, 1986b). EPA (1988a) classified tetrachloroethylene as a Group B2 carcinogen – Probable Human Carcinogen, on the basis of these studies. EPA (1985e) derived an oral cancer potency factor of 5.1×10^{-2} $(mg/kg/day)^{-1}$ based on the liver tumors observed in the NCI (1977a) gavage bioassay for mice. The inhalation cancer potency factor for tetrachloroethylene of 3.3×10^{-3} $(mg/kg/day)^{-1}$ is based on the more recent NTP (1986b) inhalation bioassay (EPA, 1988a).

EPA (1988b) also has derived an oral RfD for tetrachloroethylene based on a study by Buben and O'Flaherty (1985). In this study, liver weight to body weight ratios were significantly increased in mice and rats treated with 71 mg/kg/day tetrachloroethylene in corn oil but not in animals treated with 14 mg/kg/day; using a NOAEL of 14 mg/kg/day and applying an uncertainty factor of 1,000, an oral RfD of 1×10^{-2} mg/kg/day was derived. OSHA has set final rule limits for a time weighted average of 25 ppm (170 mg/m^3) for tetrachloroethylene (OSHA, 1989).

1,1,1-TRICHLOROETHANE

Like other chlorinated aliphatic hydrocarbons, 1,1,1-trichloroethane (1,1,1-TCA, methyl chloroform) is rapidly and completely absorbed by both the oral and pulmonary routes. Absorption through the skin is slow. 1,1,1-TCA distributes throughout the body and readily crosses the blood-brain barrier (EPA, 1984u).

The most notable toxic effects of 1,1,1-TCA in humans and animals are central nervous system depression, anaesthesia at very high concentrations, and impairment of coordination, equilibrium, and judgment at lower concentrations (350 ppm and above). Cardiovascular effects, including premature ventricular contractions, and decreased blood pressure, can result from exposure to high concentrations of 1,1,1-TCA. Fatty changes in rodent

livers following exposure by inhalation have been reported (EPA, 1985s). No adverse reproductive effects were observed in the offspring of rats or mice exposed to 1,1,1-TCA by inhalation (Schwetz et al., 1975; York et al., 1982).

Several bioassays have investigated the carcinogenic potential of 1,1,1-TCA in experimental animals. NTP (1984a) reported preliminary results of a gavage bioassay in rats and mice in which 1,1,1-TCA increased the incidence of hepatocellular carcinomas in female mice. NTP (1984a) further concluded that 1,1,1-TCA was not carcinogenic for male rats, an association between administration of the compound and increased incidences of hepatocellular carcinomas in male mice was equivocal and the study was inadequate to evaluate carcinogenicity in female rats. These results have been questioned and the study is presently being audited (Birnbaum, 1986).

EPA (1988a) calculated an oral RfD of 9×10^{-2} mg/kg/day for 1,1,1-TCA based on an inhalation study by Torkelson et al. (1958) in which groups of rats, rabbits, guinea pigs and monkeys were exposed to 1,1,1-TCA vapors. A NOAEL of 500 ppm (90 mg/kg/day) was observed in guinea pigs in this study. An uncertainty factor of 1000 was combined with the NOAEL to derive the RfD. An inhalation RfD of 0.3 mg/kg/day for 1,1,1-TCA also has been determined by EPA (1988a). A TWA of 350 ppm (1900 mg/m^3) and a STEL of 450 ppm (2450 mg/m^3) have been set by OSHA for worker health and safety (OSHA, 1989). Applied action levels for water and air have been developed by DHS for 1,1,1-TCA. These values are 0.20 mg/l and 0.13 mg/m^3, respectively (DHS, 1988c).

TRICHLOROETHYLENE

Trichloroethylene, after oral ingestion, is virtually completely absorbed. With inhalation exposure, absorption is proportional to concentration and duration of exposure. Trichloroethylene distributes among the body tissues; metabolism occurs primarily in the liver (EPA, 1985t). Trichloroethylene is a central nervous system depressant from both acute and chronic exposure. Oral exposures of human to 15 to 25 mL (21 to 35 grams) of trichloroethylene resulted in vomiting and abdominal pain, followed by

transient unconsciousness. Exposure to high doses can result in death due to respiratory and cardiac failure (EPA, 1985t). Lesser acute effects such as headache, vertigo, and visual disturbances can occur following inhalation of trichloroethylene and tremors can result following ingestion (NIOSH, 1985b). Hepatotoxicity has been reported in human and animal studies (EPA, 1985t). Transient increased liver weights resulting from exposure to trichloroethylene was reported by Kjellstrand et al. (1983). Industrial use of trichloroethylene is often associated with dermatological problems including reddening and skin burns from contact with liquid trichloroethylene and dermatitis from exposure to its vapors. These effects are usually the result of contact with concentrated solvent, however, and no effects have been reported after exposure to trichloroethylene in dilute, aqueous solutions (EPA, 1985t).

Trichloroethylene has been observed to induce increased incidences of liver tumors in mice (NCI 1976, NTP 1983) and kidney tumors in male rats (NTP 1983) following gavage exposure. Inhalation exposure has been shown to produce lung tumors in mice (Fukuda et al., 1983). EPA (1988a) classified trichloroethylene in Group B2 – Probable Human Carcinogen based on inadequate evidence in humans and sufficient evidence of carcinogenicity from animal studies. EPA (1988a) derived an oral cancer potency factor of 1.1×10^{-2} $(mg/kg/day)^{-1}$ and an inhalation cancer potency factor of 4.6×10^{-3} $(mg/kg/day)^{-1}$ based on the mouse liver tumor data in the NCI (1976b) and NTP (1983) gavage studies.

The EPA Office of Drinking Water (EPA, 1987h,j) developed an oral RfD of 7.4×10^{-3} mg/kg/day for trichloroethylene based on a study by Kimmerle and Eben (1973) in which increased liver weights were observed in rats exposed to 55 ppm trichloroethylene, 5 days/week, for 14 weeks. A time weighted average of 50 ppm (270 mg/m^3) has been promulgated by OSHA, as well as a STEL of 200 ppm (1080 mg/m^3) for the final rule limits on trichloroethylene (OSHA, 1989). Concentrations of 0.007 mg/L and 0.007 mg/m^3 have been established as applied action levels for trichloroethylene in water and air, respectively (DHS, 1988c).

VINYL CHLORIDE

Vinyl chloride is rapidly absorbed in rats following ingestion and inhalation exposure. Dermal absorption of vinyl chloride is minor (EPA, 1985f). Absorbed vinyl chloride is distributed primarily to the liver and kidney, with lower levels found in muscle, lung, fat, spleen, and brain.

At high inhalation exposure levels, workers have experienced dizziness, headaches, euphoria, and narcosis. In experimental animals, inhalation exposure to high levels of vinyl chloride can induce narcosis and death. Lower doses result in ataxia, narcosis, congestion, and edema of the lungs and hyperemia in the liver (EPA, 1985f). Chronic inhalation exposure of workers to vinyl chloride is associated with hepatotoxicity, central nervous system disturbances, pulmonary insufficiency, cardiovascular toxicity, gastrointestinal toxicity, and acro-osteolysis (EPA, 1985f). Chronic studies of experimental animals exposed to vinyl chloride by inhalation or ingestion report effects involving the liver, spleen, kidneys, hematopoietic system, and skeletal system (EPA, 1984c).

Vinyl chloride has been demonstrated to be carcinogenic in humans and laboratory animals. Occupational exposure to vinyl chloride has been associated with an increased incidence of hepatic angiosarcoma. Vinyl chloride exposure has also been implicated in brain, lung, and hemolymphopoietic cancers in humans. Animal studies in several species support the findings of epidemiological studies. Chronic inhalation and ingestion of vinyl chloride has induced cancer in the liver and in other tissues in rats and mice (IARC, 1979a). Feron et al. (1981) fed rats vinyl chloride in the diet and found that levels as low as 1.7 and 5 mg/kg/day over their lifespan induced hepatocellular carcinoma and liver angiosarcomas, as well as other adverse hepatic effects.

EPA has classified vinyl chloride in Group A -- Human Carcinogen based on adequate evidence of carcinogenicity from epidemiological studies (EPA,

1984c). EPA (1984c) reported an oral cancer potency factor of 2.3 $(mg/kg/day)^{-1}$ for vinyl chloride based on the long-term ingestion study in rats (Feron et al., 1981). Vinyl chloride doses in the experiment ranged from 0 to 14 mg/kg/day throughout the lifetime of the animals. Terminal sacrifices were made at week 135 for males and week 144 for females. A significant dose-related increase in the incidence of hepatocellular carcinoma and hepatic angiosarcoma was observed in both male and females. EPA (1987a) has also calculated an inhalation cancer potency factor for vinyl chloride of 2.95×10^{-1} $(mg/kg/day)^{-1}$. A time weighted average of 1 ppm and a 15-minute STEL of 5 ppm have been set for workplace airborne concentrations of vinyl chloride by OSHA (1989).

Ketones

ACETONE

Acetone is absorbed in humans and animals following oral or inhalation exposure (EPA, 1984d). Due to its high water solubility, acetone is widely distributed in the body. Acetone is considered to be one of the least toxic industrial solvents (Krasavage et al., 1982). Chronically exposed workers did not suffer any lasting ill effects from exposures to an airborne average concentration of 1006 ppm for 8 hours/day (Raleigh and McGee, 1970). Exposure to acetone vapors produces irritation of the mucosal membranes in humans (EPA, 1984d). Inhalation of acetone produces headaches and dizziness in humans, while direct contact may lead to dermatitis (NIOSH, 1985b). In rats, slight increases in organ weights and decreases in body weights have been observed following long-term exposure to acetone (EPA, 1986b). No information on the reproductive effects of acetone in mammals was located in the reviewed literature (EPA, 1984d; Krasavage et al., 1982; Shepard, 1986). McLaughlin et al. (1964) found no evidence of teratogenic effects after injecting acetone into the yolk sacs of fertile chick eggs.

Acetone is currently being tested for carcinogenicity in rats and mice exposed to contaminated drinking water by the National Toxicology Program.

Previous carcinogenicity bioassays are limited to skin-painting studies in which acetone served as a solvent for other compounds. In general, tumors were not induced by acetone alone in these assays.

EPA (1988a) derived an oral RfD for acetone of 0.1 mg/kg/day based on a study sponsored by the EPA Office of Solid Waste (EPA, 1986b) in which increased liver and kidney weights and nephrotoxicity were observed in rats exposed orally to acetone; an uncertainty factor of 1,000 was used to derive the RfD. OSHA has set a time weighted average and a STEL of 750 ppm (1800 mg/m^3) and 1000 ppm (2400 mg/m^3) for acetone, respectively (OSHA, 1989).

2-BUTANONE (METHYL ETHYL KETONE)

Absorption of methyl ethyl ketone from the gastrointestinal tract and from the lungs can be inferred from systemic toxic effects observed following acute oral exposure and acute and subchronic inhalation exposures (Lande et al., 1976). Inhalation of 2-butanone causes irritation of eyes and nose and headaches while ingestion is known to cause dizziness and vomiting in workers (NIOSH, 1985b). Inhaled methyl ethyl ketone produces hepatotoxicity and neurological effects in rats (Cavender et al., 1983; Takeuchi et al., 1983). Schwetz et al. (1974) reported that rats exposed to inhaled methyl ethyl ketone at concentrations of 3,000 ppm displayed retarded fetal development and teratogenic effects (acaudia, imperforate anus, and brachygnathia). Methyl ethyl ketone has not been adequately tested for carcinogenicity.

EPA (1988a) derived an oral RfD of 5×10^{-2} mg/kg/day for methyl ethyl ketone based on a study by LaBelle and Brieger (1955) in which no effects were observed in 25 rats exposed to 235 ppm (693 mg/m^3 or 46 mg/kg/day) methyl ethyl ketone for 7 hours/day, 5 days/week for 12 weeks. EPA (1987a) derived an inhalation RfD of 9×10^{-2} mg/kg/day. An uncertainty factor of 1,000 was used to calculate both oral and inhalation RfDs. Airborne concentrations of 200 ppm (590 mg/m^3) and 300 ppm (885 mg/m^3) have been considered appropriate for time weighted average and STEL, respectively, for 2-butanone (OSHA, 1989).

Applied action levels for 2-butanone have been set by CDHS for water and air. These levels are 1.8 mg/l for water and 0.25 mg/m^3 for air (DHS, 1988c).

Organochlorine pesticides

ALDRIN

Aldrin is absorbed by humans following oral, inhalation, or dermal exposure (Deichmann, 1981; Feldman and Maibach, 1974; NIOSH, 1978). It is metabolically converted to and stored as dieldrin in fatty tissues (Bann et al., 1956); therefore, the toxicities of aldrin have been considered to be the same as dieldrin (ACGIH, 1986). Both compounds affect primarily the central nervous system. Reported effects in humans following acute exposure include malaise, incoordination, headache, dizziness, gastrointestinal disturbances and major motor convulsions (NRC, 1982). No adverse effects were noted in humans chronically exposed to doses up to 3 mg/kg/day (Houk and Robinson, 1967; Hunter et al., 1969). In laboratory animals, acute exposure produces irritability, tremors, and convulsions (Heath and Vandekar, 1964); chronic exposure results in hepatic and renal toxicity (Treon and Cleveland, 1955; Walker et al., 1969; Fitzhugh et al., 1964). Aldrin has been shown to be fetotoxic and/or teratogenic in hamsters and mice (Ottolenghi et al., 1974). It had marked effects on fertility, gestation, viability, and lactation in a six-generation mouse study (Deichmann, 1972).

Data relating to the carcinogenicity of aldrin in humans are limited. Aldrin has been shown to produce liver tumors in mice following chronic oral exposure (NCI, 1978a). In contrast, chronic feeding studies with aldrin in rats have generally yielded negative results with respect to carcinogenicity (NCI, 1978a; Fitzhugh et al., 1964). EPA (1988a) classified aldrin in Group B2 – Probable Human Carcinogen, with oral and inhalation cancer potency factors of 17 (mg/kg/day)$^{-1}$. These values are based on feeding studies in mice in which increased incidences of liver carcinoma were observed (Davis, 1965, reevaluated by Reuber as cited by Epstein, 1965; NCI, 1978a).

EPA (1988a) reported an oral RfD for aldrin of 3×10^{-5} mg/kg/day based on a chronic feeding study by Fitzhugh et al. (1964) in which liver lesions were observed in rats administered 0.5 ppm (0.025 mg/kg/day) aldrin in the diet; an uncertainty factor of 1000 was used to develop the RfD. Recently, OSHA has assigned a time weighted average of 0.25 mg/m^3 to aldrin for the protection of employees working with this chemical. Aldrin has also been given a skin designation by OSHA, indicating its ability to cause adverse effects to skin or exposed sensitive membranes (OSHA, 1989).

CHLORDANE

Chlordane is absorbed through the skin, lungs, and gastrointestinal tract. As with many chlorinated hydrocarbon compounds, chlordane is retained primarily in body fat. Acute intoxication due to chlordane exposure primarily involves the central nervous system. Signs include hyperexcitability, blurred vision, irritability, confusion, vomiting, and tremors (Deichmann, 1981; EPA, 1978). Chronic effects of chlordane due to ingestion are "very small" (CAST, 1976). Growth retardation and lung and liver damage have been reported in rats fed levels of 150 and 300 ppm chlordane for 2 years (Ingle, 1952).

Chlordane has been shown to produce liver tumors in mice chronically exposed to chlordane in the diet (IARC, 1973 as reviewed by Epstein 1976; NCI, 1977d; Velsicol, 1973). EPA classified chlordane in Group B2 – Probable Human Carcinogen on the basis of these results. Oral and inhalation cancer potency factors of 1.3 $(mg/kg/day)^{-1}$ have been derived by EPA for chlordane based on the significantly increased incidence of hepatocellular tumors observed in mice in the Velsicol (1973) study.

An oral RfD of 5×10^{-5} mg/kg/day has been set by EPA (1988a) based on a chronic dietary study on rats (Velsocol Chemical Corp., 1983). Rats were fed 0, 1, or 25 ppm chlordane in the diet for 130 weeks. Liver necrosis in rats fed 1 ppm was determined to be the LOAEL; there was no NOAEL in this study. An uncertainty factor of 1,000 was used to derive the RfD. Chlordane has been

assigned a time weighted average concentration of 0.5 mg/m^3 for safety in the workplace (OSHA, 1989).

DDD, DDE, DDT

DDT and DDE are absorbed through the skin and gastrointestinal tract in humans (EPA, 1984g). In humans, DDT and its metabolites are stored primarily in adipose tissue; storage of DDT in human tissues can last up to 20 years and tissue storage of DDE can last for the lifetime of the individual (NIOSH, 1978). Acute oral exposure to DDT in humans and animals causes dizziness, confusion, tremors, convulsions, and paresthesia of the extremities. Allergic reactions in humans following dermal exposure to DDT have also been reported (EPA, 1980c). Long-term occupational exposure to DDT and DDE results in increased activity of hepatic microsomal enzymes, increased serum concentrations of LDH, SGOT, and cholesterol, decreased serum concentrations of creatinine phosphokinase, increased blood pressure, and increased frequency of miscarriages (NIOSH, 1978). Liver effects, neurological effects, immunotoxicity, reduced fertility, embryotoxicity, and fetotoxicity have also been reported in animals exposed to DDT or DDE (NIOSH, 1978; McLachlan and Dixon, 1972; Schmidt, 1973).

DDT has been shown to be carcinogenic in mice and rats at several dose levels and following different dosage regimens. The principal site of action in these studies was the liver, but an increased incidence of tumors of the lung and lymphatic system were reported in several investigations (NIOSH, 1978; Tomatis et al., 1974; NCI, 1978b). DDE also caused hepatocellular carcinomas in both sexes in B6C3F1 mice (NCI, 1978b). DDT is classified as a B2 carcinogen by the EPA Carcinogen Assessment Group.

EPA (1988a) developed an oral RfD for DDT of 5×10^{-4} mg/kg/day based on a study in which liver lesions were observed in rats fed DDT (Laug et al., 1950); an uncertainty factor of 100 was used to derive the RfD.

An oral cancer potency factor of 3.4×10^{-1} $(mg/kg/day)^{-1}$, promulgated by EPA (1988a), is based on several studies on mice and rats that reported significant increased incidence in both benign and malignant liver tumors (Turusov et al., 1973; Terracini et al., 1973; Thorpe & Walker, 1973; Tomatis and Turusov, 1975; Cabral et al., 1982; Rossi et al., 1977). OSHA has set the time weighted average of DDT at 1 mg/m^3 to ensure worker safety and health (OSHA, 1989).

ENDOSULFAN

Oral and dermal absorption of endosulfan by mammals is limited. Absorption of the beta-isomer exceeds the absorption of the alpha-isomer (EPA, 1980b). Absorption is enhanced by alcohols, oils, and emulsifiers (Maier-Bode, 1968). Substantial absorption following inhalation exposure to endosulfan is not expected to occur, due to the substance's low vapor pressure (EPA, 1980b).

Acute endosulfan poisoning in humans produces symptoms including gagging, vomiting, diarrhea, agitation, tonic-clonic convulsions, dyspnea, apnea, cyanosis, loss of consciousness, and death in some cases (Hayes, 1982). Acute exposure in animals causes hyperactivity, tremors, and convulsions followed by death (WHO, 1984). Chronic exposure produces enlarged kidneys and signs of renal tubular damage with interstitial nephritis and hepatocellular changes in rats (WHO, 1984). Diets deficient in protein are reported to increase the toxicity of technical-grade endosulfan in rats (EPA, 1980b). Adverse reproductive effects including testicular degeneration and atrophy have been reported in mice and rats following chronic exposure (EPA, 1980b).

No epidemiologic studies have been conducted to assess the carcinogenic potential of endosulfan in humans. There have been two NCI bioassays in which no statistically significant increases in tumors were reported in rats and mice exposed to technical-grade endosulfan (NCI, 1978c; EPA, 1980b). These studies are limited by the early mortality of male rats and mice produced by the chronic toxicity of endosulfan exposure (EPA, 1980b). The EPA (1987a) has

not evaluated endosulfan for evidence of its human carcinogenic potential and has not derived cancer potency factors for this compound.

EPA (1987a) developed an oral RfD of 5.0×10^{-5} mg/kg/day for endosulfan based on its chronic systemic toxicity. The RfD is based on a two-generation rat reproduction study in which kidney toxicity was demonstrated at a dietary level of 0.15 mg/kg/day (Hoeschst Aktiengesellschaft, 1984).

Endosulfan has a time weighted average concentration of 0.1 mg/m^3 which was determined by OSHA as protective of worker safety and health. OSHA has also given endosulfan a skin designation (OSHA, 1989).

ENDRIN

Endrin is absorbed through the skin and via the lungs and the gastrointestinal tract. It is apparently absorbed more readily via the lungs and gastrointestinal tract than through the skin (EPA, 1985g). Endrin is rapidly metabolized in mammals, and the metabolites are quickly eliminated.

The central nervous system appears to be the major target system for acutely administered endrin. Endrin is extremely toxic following acute exposures and may cause convulsions and death. The oral dose that produces convulsions is approximately 0.2 mg/kg body weight (Davies and Lewis, 1956); in less severe poisonings, recovery is usually rapid, and there are no permanent effects. Other effects of endrin overexposure observed in humans include headaches, dizziness, weakness, loss of appetite, nausea, vomiting, mental confusion, and dysrhythmic changes in electroencephalograms. Chronic exposure to endrin has been reported to result in enlarged liver and kidneys in rats and enlarged kidneys and hearts, convulsions, and hepatocellular changes in dogs (Treon et al., 1955). Exposure to endrin caused adverse reproductive effects in mice and hamsters. Teratogenic effects associated with endrin exposure include open eye, webbed feet, cleft lip, cleft palate, and fused ribs (Ottolenghi et al., 1974). Endrin was also shown to be fetotoxic and to produce increased resorption of embryos, reduced fetal

weight, smaller litter size, and increased fetal mortality, as well as increased maternal mortality (Good and Ware, 1969; Chernoff et al., 1979; Ottolenghi et al., 1974).

Endrin has been examined for carcinogenicity in several strains of rats and dogs. Results have been negative for most studies (Deichmann et al., 1970; NCI, 1979a; Witherup et al., 1970; Treon et al., 1955b). In contrast, Reuber (1978, 1979) reported that endrin is carcinogenic in rats based on the results of one independent study and on a reevaluation of tissue sections from the NCI rat study. The carcinogenicity of endrin has been difficult to determine because of the differences between Reuber's results and those of other investigators, as well as problems with endrin toxicity and the inadequate numbers of animals used in many studies.

EPA (1985g) derived an oral RfD of 3×10^{-4} mg/kg/day for endrin based on the study by Treon et al. (1955) in which 1 ppm endrin in the diet (equivalent to 0.045-0.12 mg/kg/day) was determined to be a NOAEL. An uncertainty factor of 100 was used in determining the RfD. A time weighted average of 0.1 mg/m^3 has been promulgated by OSHA for airborne concentrations of endrin in the workplace. Endrin has also been given a skin designation by OSHA (OSHA, 1989).

HEPTACHLOR

Heptachlor is readily absorbed from the gastrointestinal tract of rats (Mizyukova and Kurchatov, 1970). In humans, acute exposure to heptachlor results in central nervous system effects including irritability, salivation, labored respiration, muscle tremors, and convulsions (EPA, 1985q). Acute exposure of animals to heptachlor produces tremors, convulsions, paralysis, and hypothermia (EPA, 1985q). Liver damage and altered liver function have also been reported following acute exposure in animals (EPA, 1987i). Chronic inhalation or dermal exposure to heptachlor has been reported to result in anemia in humans. Wang and McMahon (1979) reported a significant increase in cerebrovascular disease in workers exposed to heptachlor for over 3 months.

Mestitzova (1967) reported marked reduction in the litter size of rats, as well as reduced lifespan of suckling rats born to dams administered heptachlor in the diet.

Heptachlor has not been associated with increased tumor incidences in occupationally exposed workers (Wang and McMahon, 1979). Several cases of leukemia have been reported following chronic inhalation or dermal exposure of humans; however in many of the reported cases, individuals were exposed to other chemicals in addition to heptachlor (EPA, 1985q, 1987i). Chronic dietary exposure has been reported to produce liver tumors in mice (NCI, 1977b; Epstein, 1976). EPA (1987i) classified heptachlor in Group B2 - Probable Human Carcinogen. EPA (1987i) derived oral and inhalation cancer potency factors of 4.5 $(mg/kg/day)^{-1}$ for heptachlor based on two carcinogenicity bioassays in mice (NCI, 1977b; Epstein, 1976). In these studies, hepatocellular carcinomas were seen in increased incidence in both sexes of mice.

An oral RfD of 5×10^{-4} mg/kg/day was also derived by EPA (1988a) based on a 2-year chronic feeding study in which rats exposed to 7 ppm heptachlor in the diet developed liver lesions (Velsicol, 1955). An uncertainty factor of 300 was used to derive the RfD. Heptachlor has also been given a time weighted average of 0.5 mg/m^3 for workplace standard concentrations. In addition, OSHA has given heptachlor a skin designation (OSHA, 1989).

BENZENE HEXACHLORIDE (BHC, LINDANE)

Technical-grade benzene hexachloride (also known as BHC or hexachlorocyclohexane) is composed mainly of the alpha (55-80%), beta (5-14%), gamma (8-15%), delta (2-16%), and epsilon (1-5%) isomers. BHC is absorbed by humans and animals following oral, inhalation, and dermal exposures (EPA, 1985i; Hayes, 1982). Absorption of the various isomers of BHC following ingestion is greater than 90% of the administered dose (Albro and Thomas, 1974). The alpha, beta, and delta-isomers of BHC primarily act as depressants of the central nervous system producing symptoms of tremors, prostration, and

flaccidity of the entire musculature. Lindane (gamma-BHC) is a stimulant causing hyperexcitability, convulsions, headache and nausea when exposed by pulmonary or ingestion routes (Hayes, 1982; EPA, 1978; Deichmann, 1981). Mental and motor retardation have been reported following dermal contact with lindane (Deichmann, 1981). All the isomers induce hepatic enzymes (Hayes, 1982). Various reproductive and developmental effects from exposure to beta- and gamma-BHC have been demonstrated in rodents (Hayes, 1982; EPA, 1985i).

Hepatocellular tumors have been observed in mice exposed to alpha- and beta-BHC in the diet (EPA, 1987a). The most tumorigenic isomer is alpha-BHC, followed by the gamma-, beta-, delta-, and epsilon-isomers (Hayes, 1982; EPA, 1985i; EPA, 1987a). EPA (1988a) classified both alpha-BHC and technical grade BHC in Group B2--Probable Human Carcinogens, and beta-BHC in Group C—Possible Human Carcinogen. The weight of evidence classification for lindane (gamma-BHC) is currently under review by EPA (1987a), although EPA (1984h) has previously classified lindane in Group B2. EPA (1988a) has estimated cancer potency factors for alpha-BHC, technical grade-BHC, and beta-BHC of 6.3, 1.8, and 1.8 $(mg/kg/day)^{-1}$, respectively, based on studies by Ito et al. (1973), Munir et al. (1983), and Thorpe and Walker (1973), respectively. These cancer potency factors apply to both oral and inhalation exposures and were derived based on the incidence of hepatic tumors in mice exposed chronically to BHC in the diet (EPA, 1988). An oral cancer potency factor of 1.3 (mg/kg/day) has been promulgated for gamma-BHC (lindane) by EPA (1988a) based on the study by Thorpe and Walker (1973) in which mice exposed to 400 ppm gamma-BHC in the diet for 110 weeks developed hepatocellular carcinomas.

An oral RfD for gamma-BHC (lindane) of 3×10^{-4} mg/kg/day has been derived by EPA (1988a) based on an unpublished study in which rats were administered gamma-BHC in the diet for 12 weeks (Zoecon Corp., 1983). In this study, liver and kidney toxicity were observed at 20 ppm (1.55 mg/kg/day) but not at 4 ppm (0.3 mg/kg/day). An uncertainty factor of 1,000 was used to derive the RfD. In addition, a concentration of 0.5 mg/m^3 has been set by OSHA as the time weighted average for lindane in the workplace. Lindane has also been listed as a skin designation chemical (OSHA, 1989).

Herbicides

In the literature search for these compounds, toxicity information was not available for the specific trademark names for some of the herbicides. In these instances, toxicity information for the components of the herbicides (e.g. bromacil is a component of Krovar I and Chlorvar) is given with the name of the trademark herbicide presented in parentheses next to the component name.

2,4-D (2,4-DICHLOROPHENOXYACETIC ACID)

2,4-D is absorbed almost completely when ingested, but only slightly after dermal contact. It is distributed within the body in decreasing concentrations to: blood, liver, kidney, heart, lungs, and spleen. 2,4-D is not metabolized to a significant degree; over 80% of excreted 2,4-D is in the unchanged form. After acute doses, 2,4-D causes hypo- and hyperexcitation of the central nervous system with persistent neurological dysfunction, muscular weakness, and convulsions. Following massive doses, ataxia, paralysis and coma may occur. Skin contact with 2,4-D may cause dermatitis. Chronic oral exposure to 2,4-D produced liver and kidney damage (Hazelton Laboratories, 1983). Decreased maternal body weight, reduced offspring weight, increased fetal mortality, and malformations have been reported as adverse reproductive effects associated with 2,4-D exposure (Hansen et al., 1971; EPA, 1986; EPA, 1985z).

2,4-D has not been extensively tested for carcinogenicity. The International Agency for Research on Cancer (IARC, 1986) concluded that there is limited evidence for the carcinogenic potential of chlorinated phenoxy-herbicides (including 2,4-D and several other compounds) in humans, but it did not specifically evaluate the evidence for 2,4-D alone. Negative results were reported in one animal cancer bioassay of orally-exposed mice (Innes, 1968). EPA has not evaluated the carcinogenic potential of 2,4-D and has not derived cancer potency factors for this compound.

An oral RfD of 1×10^{-2} mg/kg/day has been derived by EPA (1988a) based on studies conducted by Dow Chemical Co. (1983) which reported hematologic, hepatic, and renal toxicity in rats. Doses of 1, 5, 15, or 45 mg/kg/day were administered to rats in their feed for 91 days. The LOAEL and NOAEL for this study were 5 and 1 mg/kg/day respectively. An uncertainty factor of 100 was used in calculating the RfD. 2,4-D has a time weighted average concentration of 10 mg/m^3 set for workplace standards (OSHA, 1989). In addition, the DHS has set applied action levels for 2,4-D, 0.035 mg/l for water and 0.0035 mg/m^3 for air (DHS, 1988c).

BROMACIL (KROVAR I, CHLORVAR)

Bromacil is an herbicide which acts by inhibiting photosynthesis. It is the active ingredient in two herbicides, Krovar I and Chlorvar, known to have been used at NAS Alameda.

Bromacil is absorbed from the gastrointestinal tract and appears to be excreted primarily in urine (NCR, 1977). Bromacil is irritating to the eyes, nose, throat, and skin. Slight transient irritation was observed in the eyes of rabbits treated with bromacil (Grant, 1984). Distension and a stilted gait were observed after acute administration of bromacil to sheep. Gastroenteritis, liver enlargement and congestion, hemorrhages in the heart, and death were observed after four days of repeated exposure in the same study (Gosselin, 1984). No adverse reproductive effects have been reported as a result of bromacil exposure.

No teratogenic effects were observed in the offspring of New Zealand white rabbits exposed to bromacil during gestation in a 3 generation 6-litter reproduction study in rats. No adverse effects upon reproduction and lactation performance, and no pathologic changes were observed in the weanling pups (Sherman and Kaplan, 1975).

No information on the carcinogenic potential of bromacil was located in the reviewed literature. EPA has not evaluated the carcinogenicity or systemic toxicity of bromacil and it has not derived any cancer potency factors or RfDs for this compound. There has been a time weighted average promulgated for bromacil. This value of 10 mg/m^3 has been determined by OSHA in the final rule limits (OSHA, 1989).

DIURON (KROVAR I, TELVAR)

Diuron may be irritating to eyes and mucous membranes in humans, though it has been reported as non-irritating to the skin of laboratory animals (Worthing, 1983). No toxic effects were noted in dogs and rats fed diets containing 250 and 500 ppm diuron for one and two years (Verschueren, 1983). Dogs fed diets containing 24-1250 ppm diuron for two years exhibited weight loss, depressed red blood cell count, elevated liver weight and increased pigment deposition in liver cells in the highest dose group (du Pont, 1964). Mice fed diuron did not develop tumors (Hayes, 1975; Innes et al., 1968, 1969). No adverse reproductive effects were noted in a three-generation rat study other than an increased incidence of wavy rib anomaly in the offspring of rats fed 250 and 500 mg/kg Karmex (80% diuron) during days 6-15 of pregnancy (Khera et al., 1979).

EPA (1987a) promulgated an oral RfD of 2×10^{-3} mg/kg/day based on the 2-year feeding study by duPont (1964). An uncertainty factor of 300 was used in deriving the RfD to account for intra- and interspecies differences as well as for the poor quality and incompleteness of the data base. A time weighted average of 10 mg/m^3 has been promulgated for workplace air concentrations of diuron (OSHA, 1989).

MONURON (TELVAR)

Monuron is absorbed after oral exposure. After administration of 175 mg/kg for 60 days or 0.1-20.0 mg/kg for 6 months, monuron-related compounds were found mostly in the lungs with decreasing amounts in the heart, liver,

brain, and kidneys (IARC, 1976). Reports on the effects of monuron exposure in humans are limited to two incidences in which dermatitis and itching were observed (EPA, 1975). General signs of toxicity due to monuron include methemoglobinemia (cyanosis) and enlarged dark spleen. In addition to these effects, pathological examination of exposed laboratory animals has demonstrated pulmonary edema and congestion and hemorrhage in the liver and kidney (Zapp, 1955). Rats fed with 500 mg/kg/day for 10 days showed evidence of weight loss. Rats maintained for 6 weeks on a diet containing 0.5, 0.05 & 0.005% monuron had a depressed growth rate at the middle dose level (Spencer, 1982).

A 3-generation reproduction study in rats fed up to 2,500 ppm monuron showed no abnormal effects on dams or pups. At the 2,500 ppm level there was a slight reduction in the average number of pups per litter (Sherman and Culic, 1971). No teratogenic effects were noted in a screening study conducted in mice by the US Department of Health, Education, and Welfare (no doses reported) (USDHEW, 1969).

Monuron has been tested for potential carcinogenic effects in rats and mice. Male rats administered 450 mg/kg/day for 18 months developed tumors in several tissues. Tumors occurred in 14 of 50 rats; stomach tumors, intestinal tumor, liver-cell carcinomas, alveolar carcinomas, and other lung carcinomas were observed (IARC, 1976). No tumors were produced in mice exposed to monuron in the diet for 18 months (Innes et al. 1968, 1969).

EPA has not evaluated the carcinogenicity or systemic toxicity of monuron. No cancer potency factors or RfDs have been derived for this compound. No work place concentration standards have been set for monuron by OSHA.

SIMAZINE (PRINCEP)

Gastrointestinal absorption of simazine has been estimated to be 70% complete in goats and sheep administered the chemical in gelatin capsules

(Bakke and Robbins, 1968). Occupational exposure to simazine has been reported to produce contact dermatitis among workers manufacturing simazine and propazine (Yelizarove, 1977). No other adverse health effects have been noted in humans. Simazine is only slightly toxic in rats, mice and rabbits (USDA, 1984), but cattle and sheep are much more susceptible to its toxicity (Hapke, 1968). Chronic exposure to simazine has been associated with reduced food intake, blood chemistry alterations, organ weight changes, and urinalysis abnormalities in rodents; however, these effects may have been due to the decreased food intake possibly because of the unpalatability of simazine (Tai et al., 1985a,b). Chronic exposure of sheep resulted in fatty liver degeneration, renal degeneration, and brain cell death in a dose related fashion (Dshurov, 1979).

Simazine was not tumorigenic in an 18 month feeding study in mice (Innes et al., 1969); however EPA (1987d) has concluded that the study was not adequate to fully assess its carcinogenic potential. In a two-year rat study (Hazelton Laboratories, 1961), orally administered simazine caused an excess of thyroid and mammary tumors; however, EPA (1987d) has also concluded that many parameters of the findings of this study are of questionable validity.

EPA (1987d) derived an oral RfD for simazine of 5×10^{-3} mg/kg/day based on a three-generation rat study (WRC, 1965) in which the NOAEL was approximately 5 mg/kg/day; an uncertainty factor of 1,000 was used. OSHA has not yet proposed any final rule limits for airborne concentrations of simazine in the work place.

GLYPHOSATE ISOPROPYLAMINE SALT (ROUNDUP)

Absorption of glyphosate via oral exposures can be inferred from its urinary excretion (EPA, 1987q). Glyphosate is not acutely toxic following oral or dermal exposures. However, acute percutaneous exposure of rabbits to glyphosate is irritating to the skin and slightly irritating to the eyes (Worthing and Walker, 1987). The reported rat oral LD_{50} is 5,600 mg/kg and the rabbit dermal LD_{50} is >5,000 mg/kg (EPA, 1987q). Subchronic

intraperitoneal administration of glyphosate to rats has resulted in reduced body weight gain, hematological alterations, and elevated levels of serum glutamic-pyruvic transaminase and leucine-amino peptidase (EPA, 1987q). No information on the carcinogenic potential of glyphosate was found in the reviewed literature.

EPA (1987a) developed an oral RfD of 0.1 mg/kg/day for glyphosate based on a three-generation reproduction study conducted by Biodynamics. An increased incidence of renal tubular dilation in the F3b offspring was observed. The RfD was derived using the NOEL from this study of 10 mg/kg/day and an uncertainty factor of 100. No final rule limits have been set for workplace concentrations of glyphosate isopropylamine salt.

Other pesticides

DIAZINON

Diazinon is readily absorbed following ingestion (Mucke et al., 1970) and can also be absorbed dermally (Muratore et al., 1960; Hayes; 1963). Diazinon is a cholinesterase enzyme inhibitor in humans and animals. Plasma, but not red blood cell, cholinesterase activity was decreased in some men exposed to diazinon (FAO/WHO, 1967). Inhibition of red cell and plasma cholinesterase activity has been observed in dogs and rhesus monkeys exposed to diazinon; decreased brain cholinesterase activity has also been reported in exposed rats (Bruce et al., 1955; Woodard et al., 1968). Exposure typically produces a broad spectrum of effects including headache, weakness, dizziness, blurred vision, psychosis, respiratory difficulties, paralysis, convulsions, and coma (Rumack and Peterson, 1980). These effects have been noted after acute exposure in humans (Weden et al., 1984), rats (DeProspero, 1972) and sheep (Anderson et al., 1969). In addition to these effects, reticulocytopenia and a high myeloid to erythroid ratio were observed in dogs and miniature swine receiving fatal doses of diazinon. Animals exposed to high daily doses were also observed to have thickening and rupture of the gastrointestinal tract and cirrhosis (Earl et al., 1968). Increased

susceptibility to the toxic effects of diazinon was observed in mice with dietary hypercholesterolemia (Hazelette, 1984). Dietary diazinon was reported to be more toxic to female rats than to males (Davies and Holub, 1980). Adverse reproductive effects have been reported in the offspring of pregnant mice exposed orally to diazinon including significantly small adrenals, pathology of the forebrain, and behavioral defects (Cranmer et al., 1978; Spyker and Avery, 1977). No evidence of carcinogenicity has been detected in chronic exposure animal cancer bioassays in rats or mice (NCI, 1978).

EPA (1987e) developed an oral RfD for diazinon of 9×10^{-5} mg/kg/day based on a 13-week study investigating decreased cholinesterase activity in rats (Davies and Holub, 1980); an uncertainty factor of 100 was used to develop the RfD. A time weighted average concentration of 0.1 mg/m^3 has been recently promulgated by OSHA for maintaining workplace safety (OSHA, 1989).

MALATHION

Absorption of malathion occurs through dermal contact, ingestion, and inhalation (Hazleton and Holland, 1953). It is metabolized in the liver. Malathion is a mild cholinesterase inhibitor; some of the central nervous system effects due to malathion exposure are giddiness, confusion, ataxia, and slurred speech (EPA, 1978). Other acute effects include respiratory difficulties such as tightness of the chest and wheezing and ocular effects such as aching behind the eyes and blurred vision. Ingestion of malathion may induce anorexia, nausea, vomiting, abdominal cramps and diarrhea. Decreased survival, growth inhibition, and an increased incidence of ring-tail disease were reported in the offspring of rats exposed to malathion in a two-generation reproduction study (Ralow and Marton, 1961). No embryotoxicity was observed in rats after oral administration on days 6-15 of pregnancy (Khera et al., 1978). Intraperitoneal injection of rats on day 11 of pregnancy produced maternal toxicity and fetal weight reduction, but no other toxic signs (Kimbrough and Gaines, 1968). No evidence of carcinogenicity was detected in chronic exposure bioassays in mice or rats (Hazleton and Holland, 1953; NCI, 1979 b,c).

EPA has promulgated an oral RfD of 2×10^{-2} mg/kg/day for malathion based on a subchronic human feeding study in which no adverse effects were observed in subjects exposed to 16 mg malathion per day for 47 days (Moeller and Rider, 1962). Subjects ingesting 24 mg malathion daily for 56 days developed no clinical effects but were found to have depressed cholinesterase levels. An uncertainty factor of 10 was used in deriving the RfD. OSHA (1989) has determined a time weighted average concentration of 10 mg/m^3 for malathion and has also given it a skin designation.

WARFARIN

Warfarin (3-(alpha'-acetonylbenzyl)-4-hydroxycoumarin) is dermally and gastrointestinally absorbed. This compound is an anticoagulant and an antimetabolite of vitamin K. Warfarin has been shown to cause hypoprothrombinemia in humans (EPA, 1978). All symptoms occur sometime after exposure (lag time of a few days to a week). Symptoms of chronic exposure include nose bleed, bleeding gums, pallor, hematomas, and occasionally paralysis due to cerebral hemorrhage. Warfarin causes dilation and engorgement of blood vessels and an increase in capillary fragility.

EPA (1988a) has set an oral RfD of 3×10^{-4} mg/kg/day for warfarin based on human clinical studies reported by Huff (1985). Warfarin has also been given a time weighted average of 0.1 mg/m^3 by OSHA for the protection of employee health and safety (OSHA, 1989).

Other Organics

BIS(2-ETHYLHEXYL)PHTHALATE

Bis(2-ethylhexyl)phthalate, also known as di-ethylhexyl phthalate (DEHP), is readily absorbed following oral or inhalation exposure (EPA, 1980i); it is poorly absorbed through the skin (NIOSH, 1985).

Chronic exposure to relatively high concentrations of DEHP in the diet can cause retardation of growth and increased liver and kidney weights in laboratory animals (NTP, 1982a; EPA, 1980i). Reduced fetal weight and increased numbers of resorptions have been observed in rats exposed orally to DEHP (EPA, 1980i). Other reproductive effects, including testicular changes in mice and rats, have also been reported (NTP/IRLG 1982).

DEHP has been reported to be carcinogenic in rats and mice, causing increased incidences of hepatocellular carcinomas or neoplastic nodules following oral administration (NTP, 1982). EPA (1986g) classified DEHP in Group B2--Probable Human Carcinogen. An interoffice work group of EPA recently reevaluated DEHP for evidence of human carcinogenic potential and confirmed this classification (EPA, 1988a). EPA (1986g) also calculated an oral cancer potency factor for DEHP of 8.4×10^{-3} $(mg/kg/day)^{-1}$ based on data from the NTP (1982) study.

EPA (1988a) developed an oral RfD for DEHP of 0.02 mg/kg/day based on a study by Carpenter et al. (1953) in which increased liver weight was observed in female guinea pigs exposed to 19 mg/kg/day in the diet for 1 year (EPA, 1988a). To date, OSHA has not set a time weighted average for bis-(2-ethylhexyl) phthalate or any other final rule limits.

POLYCHLORINATED BIPHENYLS (PCBs)

PCBs are complex mixtures of chlorinated biphenyls. The commercial PCB mixtures that were manufactured in the United States were given the trade name of "Aroclor." Aroclors are distinguished by a four-digit number (for example, Aroclor 1260). The last two digits in the Aroclor 1200 series represent the average percentage by weight of chlorine in the product.

PCBs are readily absorbed through the gastrointestinal tract and somewhat less readily through the skin; PCBs are presumably readily absorbed from the lungs, but few data are available that experimentally define the extent of absorption after inhalation (EPA, 1985m). Dermatitis and chloracne

(a disfiguring and long-term skin disease) have been the most prominent and consistent findings in studies of occupational exposure to PCBs. Several studies examining liver function in exposed humans have reported disturbances in blood levels of liver enzymes. Reduced birth weights, slow weight gain, reduced gestational ages, and behavioral deficits in infants were reported in a study of women who had consumed PCB-contaminated fish from Lake Michigan (EPA, 1985m). For experimental animals, reproductive, hepatic, immunotoxic, and immunosuppressive effects appear to be the most sensitive endpoints of PCB toxicity in nonrodent species, and the liver appears to be the most sensitive target organ for toxicity in rodents (EPA, 1985m).

A number of studies have suggested that PCB mixtures are capable of increasing the frequency of tumors, including liver tumors, in animals exposed to the mixtures for long periods (Kimbrough et al., 1975; NCI, 1978d; Schaeffer et al., 1984; Norback and Weltman, 1985). Several studies have also suggested that PCB mixtures can act to promote or inhibit the action of other carcinogens in rats and mice (EPA, 1985m). EPA (1984n) classified PCB in Group B2 – Probable Human Carcinogen based on sufficient evidence in animal bioassays and inadequate evidence from studies in humans. The EPA Carcinogen Assessment Group (EPA, 1988a) calculated a cancer potency factor of 7.7 $(mg/kg/day)^{-1}$ for PCBs based on the incidence of hepatocellular carcinomas and adenocarcinomas in female Sprague-Dawley rats exposed to a diet containing Aroclor 1260 as reported in a study by Norback and Weltman (1985). No workplace standards have been promulgated for PCBs.

POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

PAHs occur in the environment as complex mixtures of many components with varying noncarcinogenic and carcinogenic toxic properties and potencies. Only a few components of these mixtures have been adequately characterized, and only limited information is available on the relative potencies of different compounds. The PAHs are often separated into two categories for the purposes of risk assessment: carcinogenic and noncarcinogenic PAHs.

PAH absorption following oral exposure is inferred from the demonstrated toxicity of PAHs following ingestion (EPA, 1984e). PAH absorption following inhalation exposure is inferred from the demonstrated toxicity of PAHs following inhalation (EPA, 1984e). It has been suggested that simultaneous exposure to carcinogenic PAHs such as benzo[a]pyrene and particulate matter can increase the effective dose of the compound (ATSDR, 1987b). PAHs are also absorbed following dermal exposure (Kao et al., 1985).

Acute effects from direct contact with PAHs and related materials are limited primarily to phototoxicity; the primary effect is dermatitis (NIOSH, 1977a). PAHs have also been shown to cause cytotoxicity in rapidly proliferating cells throughout the body; the hematopoietic system, lymphoid system, and testes are frequent targets (Santodonato et al., 1981). Some of the noncarcinogenic PAHs have been shown to cause systemic toxicity but these effects are generally seen only at rather high doses (Santodonato et al., 1981). Slight morphological changes in the livers and kidneys of rats have been reported following oral exposure to acenaphthene. Oral administration of naphthalene to rabbits and rats has resulted in cataract formation (EPA, 1984e). Nonneoplastic lesions are seen in animals exposed to the more potent carcinogenic PAHs only after exposure to levels well above those required to elicit a carcinogenic response.

Carcinogenic PAHs are believed to induce tumors both at the site of application and systemically. Neal and Rigdon (1967) reported that oral administration of up to 250 ppm benzo[a]pyrene for approximately 110 days induced forestomach tumors in mice. Thyssen et al. (1981) observed respiratory tract tumors in hamsters exposed to up to 9.5 mg/m^3 benzo[a]pyrene for up to 96 weeks.

Benzo[a]pyrene is representative of the carcinogenic PAHs and is classified by EPA in Group B2--Probable Human Carcinogen based on sufficient evidence of carcinogenicity from animal studies and inadequate evidence from epidemiological studies (EPA, 1984e). EPA (1984p) calculated a cancer potency factor of 11.5 $(mg/kg/day)^{-1}$ for oral exposure to carcinogenic PAHs

(specifically benzo[a]pyrene) based on the study by Neal and Rigdon (1967). EPA (1984p) calculated an inhalation cancer potency factor of 6.1 $(mg/kg/day)^{-1}$ for benzo(a)pyrene based on the study by Thyssen et al. (1981). These potency factors are currently under review based on a reevaluation of the data.

EPA's Environmental Criteria Assessment Office developed an oral RfD for chronic exposure to the noncarcinogenic PAH naphthalene of 4.1×10^{-1} mg/kg/day based on the development of ocular lesions in rats (Schmahl, 1955, as cited in EPA, 19861) and epidemiologic data on occupationally-exposed coke oven workers. An uncertainty factor of 100 was applied to the animal data in the development of the reference dose.

OSHA has promulgated a time weighted average of 0.2 mg/m^3 for coal tars which includes both carcinogenic and noncarcinogenic PAHs (OSHA, 1989). Applied action levels of 0.019 mg/L for water and 0.0019 mg/m^3 for air have been established for several noncarcinogenic PAHs by DHS. For benzo(a)pyrene, DHS has set applied action levels of 8.7×10^{-5} mg/L and 8.7×10^{-6} mg/m^3 for water and air, respectively (DHS, 1988c).

INORGANICS

ANTIMONY

Absorption of antimony via oral and inhalation exposure is low (EPA, 1980d). Humans and animals exposed orally or through inhalation to either trivalent or pentavalent forms of antimony displayed electrocardiogram (ECG) changes and myocardial lesions (EPA, 1980d). Pulmonary effects including pneumoconiosis have been observed in humans exposed by inhalation, and dermatitis has occurred in individuals exposed either orally or dermally. Oral administration of therapeutic doses in humans has been associated with nausea, vomiting, and hepatic necrosis (EPA, 1980d). A single report (Balyaeva, 1967) noted an increase in spontaneous abortions, premature births,

and gynecological problems in 318 female workers exposed to a mixture of antimony metal, antimony trioxide, and antimony pentasulfide dusts.

EPA (1988a) derived an oral RfD of 4×10^{-4} mg/kg/day for antimony based on a chronic oral study (Schroeder et al., 1970) in which rats given the metal in drinking water had altered blood glucose and blood cholesterol levels and decreased lifespans. An uncertainty factor of 1,000 was used to derive the oral RfD. A time weighted average of 0.5 mg/m^3 has recently been set by OSHA for airborne concentrations of antimony and associated compounds in the work environment (OSHA, 1989).

ARSENIC

Soluble inorganic arsenic is rapidly and almost completely absorbed from the gastrointestinal tract in rats (Coulson et al., 1935). Estimates by Coulson et al. (1935) and Ray-Bettley and O'Shea (1975) indicate that greater than 95% of ingested inorganic arsenic is absorbed by man. Absorption of inhaled arsenic, in the form of an aerosol or a dust, is dependent upon particle size. Particles smaller than 5-7 microns in diameter may deposit deep in the lungs and be absorbed by the respiratory epithelium. Larger particles are deposited primarily in the upper airways, cleared from the lung by retrociliary action, and swallowed (EPA, 1984q). Once absorbed, arsenic is widely distributed. Acute exposure of humans to arsenic has been associated with gastrointestinal effects, hemolysis, and neuropathy. Respiratory irritation may occur following contact with arsenic (NIOSH, 1985b). Chronic exposure of humans to this metal can produce toxic effects on both the peripheral and central nervous systems, keratosis, hyperpigmentation, precancerous dermal lesions, and cardiovascular damage (EPA, 1984r). Arsenic is embryotoxic, fetotoxic, and teratogenic in several animal species (EPA, 1984r).

Arsenic is a known human carcinogen. Epidemiological studies of workers in smelters and in plants manufacturing arsenical pesticides have shown that inhalation of arsenic is strongly associated with lung cancer and perhaps with

hepatic angiosarcoma (EPA, 1984q,r). Ingestion of arsenic has been linked to a form of skin cancer and more recently to bladder, liver, and lung cancer (Tseng et al., 1968; Chen et al., 1986).

EPA (1987a) has classified arsenic in Group A -- Human Carcinogen, and has developed inhalation and oral cancer potency factors of 50.1 $(mg/kg/day)^{-1}$ and 1.75 $(mg/kg/day)^{-1}$, respectively. The inhalation potency factor is the geometric mean value of potency factors derived from four occupational exposure studies on two different exposure populations (EPA, 1984q). The oral cancer potency factor was based on an epidemiological study in Taiwan which indicated an increased incidence of skin cancer in individuals exposed to arsenic in drinking water (EPA, 1984q). The increase in internal cancers recently associated with arsenic exposure is under active review by EPA. Arsenic has a time weighted average of 0.5 mg/m^3 derived as a concentration acceptable for the workplace; this standard was set by OSHA as part of the final rule limit (OSHA, 1989).

ASBESTOS

Asbestos is a generic term referring to a family of naturally occurring silicates having a fibrous crystalline structure. There are six fibrous silicates defined as asbestos types: chrysotile, actinolite, cunningtonite-grunerite or amosite, anthophyllite, crocidolite, and tremolite. In humans, the primary routes of exposure to asbestos fibers are inhalation and direct ingestion or indirect ingestion following inhalation.

The primary noncarcinogenic health effect of asbestos is asbestosis, a chronic lung disease associated with functional disabilities and early mortality; however, development of asbestosis is usually associated only with high-level occupational exposure (EPA, 1986h). For low-level environmental exposure, cancer is considered a more appropriate endpoint for criteria development than asbestosis.

Deposition and absorption of asbestos fibers can be influenced by fiber characteristics such as fiber length, fiber diameter, aspect ratio (ratio of length to diameter), fiber number, stability of fibers in the body, surface chemistry of the fiber, interactions between fibers and other surfaces, fiber translocation and migration, overall fiber dose, and fiber type (Schneiderman et al., 1981). Specific data relating individual asbestos type and physical characteristics of the fiber with biological activity via ingestion are lacking. Following inhalation, there is some evidence to suggest a relationship between asbestos fiber dimension and carcinogenic potential. Long, thin fibers (> 5 microns in length, aspect ratio >3) appear to elicit the greatest biological response. However, a critical fiber length below which there would be no carcinogenic activity has not been demonstrated. Fibers less than 5 microns in length appear to be capable of producing mesothelioma (OSHA, 1986; EPA, 1986h), and the results of one analysis show that carcinogenicity appears to be a continuously increasing function of the aspect ratio (Bertrand and Pezerat, 1980). A reanalysis of Stanton's original data (Wylie et al., 1988) concludes that factors other than size and shape may play a role in asbestos carcinogenicity.

The carcinogenicity of asbestos following ingestion has not been conclusively demonstrated by direct studies. In an NTP (1984) bioassay in male rats, a significant increase in benign epithelial neoplasms in the large intestine was interpreted as limited evidence that orally ingested chrysotile fibers may be carcinogenic (EPA, 1985b). Available data from occupational studies also suggest a link between inhalation and subsequent ingestion of asbestos and gastrointestinal cancer (EPA, 1986h). Inhalation exposure in humans and experimental animals can result in both lung cancer and mesothelioma (EPA, 1986h).

EPA (1985ab) developed an oral unit risk factor of 1.4×10^{-13} $(\text{fiber/liter})^{-1}$ based on the NTP (1984) bioassay in which benign neoplasms were observed in male rats exposed to asbestos (>10 microns in length) in drinking water; this cancer potency factor was used by EPA as the basis for the drinking water maximum contaminant level goal. There are a number of

uncertainties associated with this approach including the absence of adequate dose-response data from human populations exposed via ingestion, the induction of benign tumors only, and that the criterion is limited to fibers greater than 10 microns in length. No oral cancer potency factor has been derived for any environmental medium other than drinking water in which asbestos concentration may be reported as fiber mass per unit volume ($\mu g/m^3$) rather than fibers per unit volume.

EPA (1988a) derived an inhalation cancer potency factor for asbestos of 2.3×10^{-1} $(mg/kg/day)^{-1}$ based on epidemiologic and animal data, and based on the evidence of its carcinogenicity, asbestos has been classified in Group A - Human Carcinogen by the EPA's Cancer Assessment Group.

The airborne concentrations of asbestos determined by OSHA to be acceptable for a time weighted average is 0.2 fiber/cubic centimeter of air for an 8-hour work day (OSHA, 1989).

BARIUM

Adverse effects in humans following oral exposure to soluble barium compounds include gastroenteritis, muscular paralysis, hypertension, ventricular fibrillation, and central nervous system damage (EPA, 1984i; Perry et al., 1983). Inhalation of barium sulfate or barium carbonate in occupationally exposed workers has been associated with baritosis, a benign pneumoconiosis (Goyer, 1986). Inhalation of barium compounds may cause upper respiratory tract irritation, ingestion may cause muscle spasms and a slowing of pulse. Eye irritation and skin burns may also result from contact with barium (NIOSH, 1985b). Rats exposed chronically to barium in drinking water developed increased blood pressure. No increase in blood pressure was seen in humans exposed to elevated concentrations of barium in drinking water (EPA, 1984i). Inhalation of barium carbonate dust by experimental animals has been associated with reduced sperm count, increased fetal mortality, and atresia of the ovarian follicles (EPA, 1984i). Data regarding the carcinogenic potential of barium are limited. One study reported no increase in tumor incidence in

either rats or mice exposed to 5 mg/liter barium acetate in drinking water throughout the lifespan (EPA, 1984i).

EPA (1988a) derived an oral RfD based on a chronic rat study in which a lowest-observed-adverse-effect level (LOAEL) for increased blood pressure was observed (Perry et al., 1983). Using the LOAEL of 5.1 mg/kg/day and an uncertainty factor of 100, an oral RfD of 5×10^{-2} mg/kg/day was calculated. EPA (1988b) has also developed an inhalation RfD of 1.4×10^{-4} mg/kg/day for barium based on a study by Tarasenko et al. (1977). In this study, rats were exposed to barium carbonate dust at airborne concentrations of up to 5.2 mg/m^3 for 4-6 months. Adverse effects noted at this concentration included decreased body weight, alterations in liver function, and increased fetal mortality. An uncertainty factor of 1,000 was used in developing the RfD. Average airborne concentrations of 0.5 mg/m^3 have been determined by OSHA to be protective of worker health and safety for an 8-hour work day (OSHA, 1989). Applied action levels of 0.35 mg/l and 0.0049 mg/m^3 have been developed by DHS for acceptable water and air concentrations (DHS, 1988).

BERYLLIUM

Beryllium is not well absorbed following exposure by any route. Body concentrations of beryllium have been reported to be highest in bone, liver, and kidney (EPA, 1986c). Dermal exposure to soluble beryllium compounds can cause contact dermatitis, ulceration, and granulomas (Hammond and Beliles, 1980). Acute respiratory effects of beryllium include pharyngitis, rhinitis, tracheobronchitis, and acute pneumonitis. Chronic beryllium exposure may result in lung inflammation, coughing, chest pain, and general weakness. Chronic skin lesions may appear after a long latent period in conjunction with the pulmonary effects (Hammond and Beliles, 1980). Beryllium has not been reported to cause adverse reproductive effects (Shepard, 1986; Barlow and Sullivan, 1982).

Epidemiological studies suggest that inhalation exposure to beryllium may result in an increased risk of lung cancer. The International Agency for

Research on Cancer (IARC) has concluded that beryllium is probably carcinogenic to humans, based on an overall consideration of the available data (IARC, 1982). The EPA Carcinogen Assessment Group (CAG) has classified beryllium in Group B2—Probable Human Carcinogen for inhalation exposure (EPA, 1986). EPA derived an inhalation cancer potency factor of 8.4 $(mg/kg/day)^{-1}$ for exposure to beryllium, based on data from an epidemiological study by Wagoner et al. (1980), and industrial hygiene reviews by NIOSH (1972) and Eisenbud and Lisson (1983).

EPA (1986c) also established an oral RfD of 5×10^{-3} mg/kg/day for beryllium based on a chronic study in which no adverse effects were seen in rats exposed to 0 or 5 ppm beryllium sulfate in the drinking water for a lifetime (Schroeder and Mitchner, 1975). An uncertainty factor of 100 was included in the RfD. OSHA has promulgated a time weighted average of 0.002 ppm as an acceptable workplace airborne concentration standard. In addition a STEL of 0.005 ppm and a ceiling limit of 0.025 ppm have been set by OSHA in the final rule limits (OSHA, 1989).

CADMIUM

Gastrointestinal absorption of cadmium in humans ranges from 5-6% (EPA, 1985j). Pulmonary absorption of cadmium in humans is reported to range from 10% to 50% (CDHS 1986). Cadmium bioaccumulates in humans, particularly in the kidney and liver (EPA, 1985j,k). Acute exposure to cadmium may cause coughing, tightening of the chest, headache, chills, and muscle aching following ingestion or inhalation (NIOSH, 1985b). Chronic oral or inhalation exposure of humans to cadmium has been associated with renal dysfunction, itai-itai disease (bone damage), hypertension, anemia, endocrine alterations, and immunosuppression. Renal toxicity occurs in humans at a cadmium concentration of 200 $\mu g/g$ in the renal cortex (EPA, 1985j). Cadmium is a well-documented animal teratogen (EPA, 1985j).

Epidemiological studies have demonstrated a strong association between inhalation exposure to cadmium and cancers of the lungs, kidney, and prostate

(EPA, 1985j). In experimental animals, cadmium induces injection-site sarcomas and testicular tumors. Cadmium has not been shown to be carcinogenic following oral exposures in humans or animals. When administered by inhalation, cadmium chloride is a potent pulmonary carcinogen in rats. EPA (1988a) classified cadmium as a Group B1 carcinogen (Probable Human Carcinogen). This classification applies to agents for which there is limited evidence of carcinogenicity in humans from epidemiologic studies combined with sufficient evidence in experimental animals. EPA (1985j) derived an inhalation cancer potency factor of 6.1 $(mg/kg/day)^{-1}$ for cadmium based on an epidemiologic study by Thun et al. (1985).

EPA (1988b) has derived two separate RfD's using renal toxicity as an endpoint, and a safety factor of 10. The RfD associated with oral exposure to cadmium in drinking water is 5×10^{-4} mg/kg/day, and is based upon the LOAEL of .005 mg/kg identified in humans. The RfD associated with oral exposure to cadmium in food, or other nonaqueous oral exposures is 1×10^{-3} mg/kg/day. Ceilings for cadmium fumes and dust in the workplace have been set by OSHA at 0.3 mg/m^3 and 0.6 mg/m^3, respectively (1989).

CHROMIUM

Chromium is an essential micronutrient and is not toxic in trace quantities (EPA, 1980e). Chromium exists principally in two states, as chromium (III) and chromium (VI). Following oral exposure, absorption of chromium (III) is low (<1%) while absorption of chromium (VI) is approximately 10% (ATSDR, 1987a). Acute oral exposure to high levels of soluble chromium (VI) and chromium (III) can produce kidney and liver damage; the target organs affected by chronic oral exposure remain unidentified (EPA, 1984j). Chronic inhalation exposure may cause respiratory system damage (EPA, 1984j). Chromium VI compounds are strong oxidizing agents and may produce irritant effects (EPA, 1984j). Certain chromium salts have been shown to be teratogenic and embryotoxic in mice and hamsters following intravenous or intraperitoneal injection (EPA, 1984j).

Epidemiological studies of worker populations have clearly established that inhaled chromium (VI) is a human carcinogen; the respiratory passages and the lungs are the target organs (EPA, 1984j). Inhalation of chromium (III) and ingestion of chromium (VI) or (III) have not been associated with carcinogenicity in humans or experimental animals (EPA, 1984j). EPA has classified inhaled chromium (VI) in Group A – Human Carcinogen (EPA, 1988a); inhaled chromium (III) and ingested chromium (III) and (VI) have not been classified with respect to carcinogenicity. EPA (1988) developed an inhalation cancer potency factor of 41 $(mg/kg/day)^{-1}$ for chromium (VI) based on an increased incidence of lung cancer in workers exposed to chromium over a 6 year period, and followed for approximately 40 years (Mancuso, 1975).

EPA (1988a) derived an oral RfD of 5×10^{-3} mg/kg/day for chromium (VI) based on a study by MacKenzie et al. (1958) in which no observable adverse effects were observed in rats exposed to chromium (VI) in drinking water for 1 year. EPA (1988a) also developed an RfD of 1 mg/kg/day for chromium (III) based on a study in which rats were exposed to chromic oxide baked in bread; no effects due to chromic oxide treatment were observed at any dose level (Ivankovic and Preussman, 1975). An uncertainty factor of 100 was used to calculate the RfD. Chromium III has been given a time weighted average concentration of 0.5 mg/m^3 by OSHA for the workplace standard (OSHA, 1989).

COBALT

Cobalt is an essential trace element in human nutrition. Cobalt is generally well absorbed following ingestion. Acute ingestion of large doses produces gastrointestinal disturbances (vomiting, diarrhea), and a sensation of warmth and coughing (Hammond and Beliles, 1980; NIOSH, 1985b). Chronic oral exposure to cobalt in high doses can cause goiter, decreased thyroid function, increased heart and respiratory rates, and blood lipid changes (Hammond and Beliles, 1980). Chronic exposure to cobalt dust has been reported to produce respiratory disease in workers (Stokinger, 1981). Cobalt salts included in a beer formulation at concentrations of 1.2 to 1.5 mg/liter were reported to be responsible for a number of deaths due to congestive heart

failure (NAS, 1977). Cobalt administered to laboratory rodents produced adverse reproductive effects including craniofacial developmental abnormalities in mice (Leonard et al., 1984) and decreased body weight in rats (Shepard, 1986).

Cobalt has been reported to cause sarcomas at the site of injection in rats (Gilman, 1962; Heath, 1960); however the results of carcinogenesis studies performed by other routes of exposure have been negative. EPA has not classified cobalt on the basis of its carcinogenicity. No cancer potency factors or RfDs have been derived for this element. Recently OSHA promulgated a time weighted average of 0.005 mg/m^3 for cobalt concentrations in the workplace (OSHA, 1989).

COPPER

Copper is an essential element. A daily copper intake of 2 mg is considered to be adequate for normal health and nutrition; the minimum daily requirement is 10 $\mu g/kg$ (EPA, 1985u). Adverse effects in humans resulting from acute overexposure to copper by ingestion include salivation, gastrointestinal irritation, nausea, vomiting, hemorrhagic gastritis, and diarrhea (ACGIH, 1986). Dermal or ocular exposure of humans to copper salts can produce irritation of mucous membranes (ACGIH, 1986). Acute inhalation of dusts or mists of copper salts by humans may produce irritation of the mucous membranes and pharynx, ulceration of the nasal septum, and metal fume fever. The latter condition is characterized by chills, fever, headache, and muscle pain. Limited data are available on the chronic toxicity of copper; however, chronic overexposure to copper by humans has been associated with anemia (ACGIH, 1986). Results of several animal bioassays suggest that copper compounds are not carcinogenic by oral administration; however, some copper compounds can induce injection-site tumors in mice (EPA, 1985u).

EPA (1988a) has reported an oral RfD 3.7×10^{-2} mg/kg/day based on the observation that 5.3 mg/day represents a human LOAEL and that doses higher than this may cause gastrointestinal disturbances and other acute toxic

effects. An uncertainty factor of 2 was included in the calculation of the RfD. A time weighted average concentration of 0.1 mg/m^3 has been promulgated by OSHA for copper in the workplace (OSHA, 1989).

CYANIDES

Cyanides are readily absorbed from the gastrointestinal tract, lungs, and skin and, once absorbed, are rapidly distributed throughout the body (EPA, 19851). The toxic effects in humans following acute oral exposure to cyanides include hyperventilation, vomiting, unconsciousness, convulsions, vascular collapse, cyanosis, and death (EPA, 19851). Inhalation of high concentrations of hydrogen cyanide (HCN) gas results in almost immediate collapse, respiratory arrest, and death within minutes (DiPalma, 1971). Airborne hydrogen cyanide concentrations between 99 and 528 mg/m^3 are fatal within 30-60 minutes (NIOSH, 1976). There are limited data on chronic exposures to cyanide in humans, although the following effects have been identified in chronically exposed workers in some epidemiologic studies: neurological dysfunction, lacrimation, abdominal pain, muscular weakness, and shortness of breath (NIOSH, 1976). Cyanide can cause teratogenic effects when subcutaneously administered to hamsters; this teratogenic effect has not been observed in other species although some reproductive toxicity has been noted (EPA, 19851).

EPA (1988a) calculated an RfD for cyanide based on a study by Howard and Hanzal (1955) in which rats were maintained for 2 years on a diet fumigated with hydrogen cyanide. No adverse effects were noted at the highest dose administered (10.8 mg/kg/day). Using a NOAEL of 10.8 mg/kg/day and an uncertainty factor of 500, an oral RfD of 0.02 mg/kg/day was derived (EPA, 1988a). OSHA has set the time weighted average of cyanide at 5 mg/m^3 (OSHA, 1989).

IRON

Gastrointestinal absorption of iron in humans ranges from 1 to 25% (EPA, 1984k). Absorption of iron following inhalation exposure has not been thoroughly studied. Iron is an essential element and is nontoxic at doses necessary for maintaining normal health and nutrition (EPA, 1984k). However, overexposure to iron can cause adverse health effects. Gastrointestinal irritation is the primary effect observed in humans following acute oral overexposure to iron. Chronic oral overexposure of humans has been associated with gastrointestinal bleeding, metabolic acidosis, hepatic toxicity, hemosiderosis, and hemochromatosis (EPA, 1984k). Human fatalities have occurred following ingestion of iron at doses of 100 mg/kg/day (Venugopal and Luckey, 1978). Chronic inhalation overexposure of humans to iron-containing dusts and fumes produces respiratory irritation and various pulmonary lesions (EPA, 1984k). There is limited evidence from studies with experimental animals that certain soluble iron salts are teratogenic. Certain iron compounds are also reported to be genotoxic.

Iron oxide enhances the carcinogenic action of various organic carcinogens (benzo[a]pyrene for example) and may act as a tumor promoter. Local sarcomas have been induced by subcutaneous injection of iron-dextran (EPA, 1984k). EPA (1984k) has placed iron in Group C – Possible Human Carcinogen. However, no quantitative estimate of cancer potency has been derived for iron.

EPA has not derived oral or inhalation RfDs for iron. The National Research Council of the National Academy of Sciences (NRC, 1980) has noted that the levels of iron associated with the long-term toxicity in monogastric animals are 340 to 1,700 times greater than the dietary requirement. Therefore, the maximum recommended daily intake of iron could be used as a conservative allowable intake for chronic oral exposure. The NRC (1980) has recommended daily dietary allowances (RDAs) for iron of between 10 and 60 mg (0.1-1 mg/kg/day). The soluble salts of iron have been given a time weighted

average of 1 mg/m^3 as a safe airborne concentration for employees working with these compounds (OSHA, 1989).

LEAD

Absorption of lead from the gastrointestinal tract of humans is estimated at 10%-15%. For adult humans, the deposition rate of particulate airborne lead is 30%-50%, and essentially all of the lead deposited is absorbed. Lead is stored in the body in bone, kidney, and liver (EPA, 19841). The major adverse effects in humans caused by lead include alterations in the hematopoietic and nervous systems. The toxic effects are generally related to the concentration of this metal in blood. Blood concentration levels of over 80 $\mu g/dl$ in children and over 100 $\mu g/dl$ in sensitive adults can cause severe, irreversible brain damage, encephalopathy, and possible death. Lower blood concentrations of lead (30-40 $\mu g/dl$) have been associated in humans with altered nerve conduction, altered testicular function, renal dysfunction, and anemia. Even lower blood lead concentrations (<10-20 $\mu g/dl$) have been associated with subtle deficits in learning ability. Lead exposure also has been associated with reproductive effects in humans including spontaneous abortions, premature delivery, and early membrane rupture; however, reliable exposure estimates are lacking in these cases. Decreased fertility, fetotoxic effects, and skeletal malformations have been observed in experimental animals exposed to lead (EPA, 19841).

Oral ingestion of certain lead salts (lead acetate, lead phosphate, lead subacetate) has been associated with increased renal tumors in experimental animals, but doses of lead that induced kidney tumors were high and were beyond the lethal dose in humans (EPA, 1985v). EPA (1985v) classified these lead salts as Group B2 Carcinogens--Probable Human Carcinogens. This category applies to those agents for which there is sufficient evidence of carcinogenicity in animals and inadequate evidence of carcinogenicity in humans. EPA (1985v) also noted that the available data provide an insufficient basis on which to regulate these compounds as human carcinogens.

EPA (1988a) also considered it inappropriate to develop an RfD for inorganic lead and lead compounds, since no dose threshold can be identified for many of the health effects associated with lead intake. There is currently no promulgated time weighted average or other final rule limits for lead as they are under court remand (OSHA, 1989).

MANGANESE

Manganese is absorbed at low levels following oral or inhalation exposure (EPA, 1984v). Metal fume fever, dry throat, coughing, and tightening of the chest are all short term effects of exposure to manganese (NIOSH, 1985b). Chronic oral and inhalation exposure of humans to manganese results in a condition known as manganism, a progressive neurological disease characterized by speech disturbances, tremors, and difficulties in walking. Altered hematologic parameters (hemoglobin concentrations, erythrocyte counts) have also been observed in persons exposed chronically. Manganese has not been reported to be teratogenic; however, it has been reported to cause depressed reproductive performance and reduced fertility in humans and experimental animals. There is no evidence that manganese is carcinogenic.

EPA (1984v) established an oral RfD of 2.1×10^{-1} mg/kg/day based on studies in which no adverse effects were observed in rats exposed to 1 mg/ml (21 mg/kg/day) in drinking water (Lai et al., 1982; Leung et al., 1981). EPA (1984v) also established an inhalation RfD of 3×10^{-4} mg/kg/day based on human epidemiological data which suggests that the exposure threshold for toxic effects is approximately 300 $\mu g/m^3$ (2/μg/day). Uncertainty factors of 100 were used in deriving both RfDs. A ceiling limit of 5 mg/m^3 has been promulgated by OSHA (1989) for work places using manganese compounds.

MERCURY

Elemental and inorganic mercury are poorly absorbed from the gastrointestinal tract (less than 15%) and easily absorbed by inhalation (approximately 80%) in humans. Organic mercury is almost completely absorbed

from the gastrointestinal tract and is assumed to be well absorbed via inhalation (EPA, 1984m). The extent of dermal absorption is not precisely known, but alkyl mercury is probably well absorbed. The toxicity of mercury depends to some extent on its chemical form. Irrespective of the chemical form, the major target organs for mercury toxicity are the central nervous system (CNS) and the kidney. Inorganic and organic mercury compounds can cause somewhat different neurotoxic effects initially, although both will elicit the same effects at higher doses (Hammond and Beliles, 1980). Organic mercury compounds are generally more neurotoxic than inorganic mercury.

Classical symptoms of elemental mercury vapor intoxication are mental disturbances, objective tremors, and gingivitis, which have been observed following chronic occupational exposure to average air concentrations of 0.1-0.2 mg/m^3 mercury (EPA, 1984m). The CNS appears to be the primary target of organic mercury intoxication. Miettinen (1973) estimated that an intake of 200 μg/day of organic mercury corresponded to a blood level of 200 ng/ml blood, which was estimated to be a threshold level for the development of neurological symptoms (EPA, 1984m). Clinical symptoms including paresthesia, loss of sensation in the extremities, ataxia, constriction of the visual field, and hearing impairment suggest damage to peripheral nerves, but histopathological documentation is lacking (WHO, 1976). CNS lesions similar to those in humans, proteinuria, and morphological kidney changes have been reported in animals exposed to mercury (Koller, 1979; EPA, 1987f). Chronic low dose industrial exposure has been shown to result in proteinuria. Methyl mercury does not appear to be nephrotoxic (Hammond and Beliles, 1980). Several investigators have reported embryotoxic and teratogenic effects in experimental animals treated with organic mercury. The most common findings are neurological effects, but skeletal malformations including cleft palate and jaw and facial defects have been reported in mice, hamsters and dogs. Brain damage, but not anatomical defects, has been reported in humans exposed prenatally to organic mercury (EPA, 1984m).

Limited data are available regarding the carcinogenic potential of mercury in humans or animals. Methyl mercury chloride has been shown to

induce kidney tumors in mice in one test (Mitsumori et al., 1987), but other tests with organic and inorganic mercury have generally been negative. EPA has not evaluated the carcinogenic potential of mercury and no cancer potency factors have been derived for these compounds.

EPA (1986f) has derived an oral RfD for inorganic mercury of 0.002 mg/kg/day based on a study in which rats were exposed to mercury (as mercuric acetate) in the diet (Fitzhugh et al., 1950). A LOAEL of 2 mg/kg/day was identified based on the presence of morphological changes in the kidney and an uncertainty factor of 1,000 was used to derive the RfD (EPA, 1986f).

An RfD for methyl mercury of 0.0003 mg/kg/day was developed by EPA based on several studies investigating central nervous system effects in humans exposed to mercury. A blood level of 200 ng mercury/ml of blood was identified as the LOAEL from these studies. An uncertainty factor of 10 was applied in calculating the RfD. The earliest detected effects were CNS effects such as ataxia and paresthesia (EPA, 1986f). Organic mercury has a time weighted average of 0.01 mg/m^3 and a STEL of 0.03 mg/m^3 promulgated by OSHA. A ceiling limit of 0.1 mg/m^3 has been promulgated for inorganic mercury. Skin designations have been assigned for both organic and inorganic mercury (OSHA, 1989). DHS has derived applied action levels of 0.002 mg/l in water and 0.00007 mg/m^3 in air for inorganic mercury (DHS, 1988c).

NICKEL

Nickel compounds can be absorbed following inhalation, ingestion, or dermal exposure. The amount absorbed depends on the dose administered and the chemical and physical form of the particular nickel compound (EPA, 1986d). Adverse effects associated with acute exposure in animals have included depressed weight gain, altered hematological parameters, and increased iron deposition in blood, heart, liver, and testes (EPA, 1987g). Chronic or subchronic exposures of experimental animals to nickel have been associated with reduced weight gain, degenerative lesions of the male reproductive tract, asthma, nasal septal perforations, rhinitis, sinusitis, hyperglycemia,

decreased prolactin levels, decreased iodine uptake, and vasoconstriction of the coronary vessels. Dermal exposure of humans to nickel produces allergic contact dermatitis (EPA, 1986d). Teratogenic and fetotoxic effects have been observed in the offspring of exposed animals (EPA, 1986d).

Epidemiological evidence indicates that inhalation of nickel refinery dust and nickel subsulfide is associated with cancers of the nasal cavity, lung, larynx, kidney, and prostate (EPA, 1986d). Inhalation exposure of experimental animals to nickel carbonyl or nickel subsulfide induces pulmonary tumors (EPA, 1986d). Several nickel salts cause localized tumors when administered by subcutaneous injection or implantation. EPA (1987g) categorized nickel refinery dust and nickel subsulfide by inhalation in Group A--Human Carcinogens. EPA (1987g) derived cancer potency factors of 0.84 $(mg/kg/day)^{-1}$ and 1.7 $(mg/kg/day)^{-1}$, for these two compounds, respectively, on the basis of epidemiological studies of nickel refinery workers. Nickel carbonyl by inhalation is categorized in Group B2--Probable Human Carcinogen; however, a potency factor has not been derived for this compound (EPA, 1986d).

EPA (1988a) derived an oral RfD for nickel of 2×10^{-2} mg/kg/day based on a study by Ambrose et al. (1976) in which rats administered 100 ppm nickel in the diet for 2 years did not experience decreased weight gain. OSHA (1989) has promulgated a time weighted average of 0.1 mg/m^3 for nickel. Applied action levels for nickel in water and air have been set by DHS at 0.4 mg/l and 0.0001 mg/m^3, respectively (DHS, 1988c).

SELENIUM

Selenium is an essential element in animals and probably in humans (EPA, 1980g, 1984y). Absorption of selenium from the gastrointestinal tract is generally high, but is dependent upon many factors including dietary intake level and the chemical nature of the compound. Data on other routes of absorption are limited, but absorption via the lungs has been suggested in at least one study (EPA, 1984y). Acute effects of selenium exposure following ingestion include digestive tract hemorrhage, degeneration of the myocardium,

liver and kidney and brain damage. Eye, nose, and throat irritation may also occur following inhalation exposure in addition to visual disturbance, headache, chills, and fever (EPA, 1984y; NIOSH, 1985). Signs of chronic selenium exposure are depression, nervousness, dermatitis, and gastrointestinal disturbances. Occupational exposure to selenium has been reported to result in respiratory and gastrointestinal irritation, cold-like symptoms, and metallic taste in the mouth (EPA, 1984y). Adverse reproductive effects including failure to breed and increased perinatal death have been observed in animals (EPA, 1984y). Several reports suggest that selenium may be a teratogen in humans, but this has not been conclusively proven (EPA, 1984y).

There are no epidemiological studies that suggest that selenium may be carcinogenic in humans (EPA, 1984y; IARC, 1975). However, a few studies have suggested that certain selenium compounds may cause liver tumors in laboratory animals. EPA has not classified selenium as to its carcinogenic potential and no cancer potency factors have been calculated for this compound.

EPA (1984y) calculated an oral RfD of 3×10^{-3} mg/kg/day based on an epidemiological study reported by Yang et al. (1983) in which an LOAEL of 3.2 mg/day was identified. An uncertainty factor of 15 was used to derive the RfD. EPA (1984y) also derived an inhalation RfD of 1×10^{-3} mg/kg/day based on a study of occupationally exposed workers (Glover, 1967); an uncertainty factory of 10 was included in the calculation. Selenium has been given an 8-hour time weighted average concentration of 0.2 mg/m^3 to protect employee health in the workplace (OSHA, 1989).

SILVER

Silver in various forms is absorbed to a limited extent following oral and inhalation exposures (EPA, 1985y). The acute toxic effects in humans following oral exposure to silver include corrosive damage to the gastrointestinal tract leading to shock, convulsions, and death. Direct contact with silver may cause skin irritation (NIOSH, 1985b). In animals,

acute exposure has been shown to affect the central nervous system and to cause respiratory paralysis (Hill and Pillsbury, 1939). The primary effect of silver in humans following chronic exposure, is argyria, a permanent bluish-metallic discoloration of the skin and mucous membranes, which can be either localized or generalized. Silver also accumulates in the blood vessels and connective tissue (EPA 1980). No information on the adverse reproductive or teratogenic effects of silver was located in the reviewed literature.

Silver was used in the past as a therapeutic agent in humans and no evidence of carcinogenicity has been reported to be associated with this use. In laboratory animals, injection of colloidal silver has been reported to induce tumors at the site of injection (Schmahl and Steinhaff, 1960). No tumors were observed in rats given intramuscular injections of silver powder for six months (Furst and Schlauder, 1977).

EPA (1988) derived an oral RfD of 0.003 mg/kg/day for silver based on the human case reports of Gaul and Staud (1935), Blumberg and Carey (1934), and East et al. (1980). In these studies, argyria was observed at an average dose of silver of 0.0052 mg/kg/day; an uncertainty factor of 2 was used to derive the RfD. OSHA (1989) has promulgated a time weighted average of 0.01 mg/m^3 for airborne silver in the workplace. An applied action level for water of 0.7 mg/l has been established by DHS, in addition to an applied action level for air of 0.007 mg/m^3 (DHS, 1988c).

THALLIUM

Following acute poisoning, the highest thallium concentrations are found in the kidneys with lesser amounts found in intestines, thyroid gland, testes, pancreas, skin, bone, and spleen. Large amounts are excreted in urine within the first 24 hours following the initial exposure to thallium.

Thallium is acutely toxic regardless of the chemical form of the compound or route of administration. Since 1932, hundreds of cases of thallotoxicosis due to ingestion of thallium-based pesticides have been

reported (ACGIH, 1986). Of the children poisoned by thallium ingestion who survived and were later examined, mental retardation and psychoses were the most common findings (ACGIH, 1986). The effects of thallium toxicity are similar in humans and animals. The most commonly noted response to thallium exposure is alopecia, but neurological and gastrointestinal findings are frequently found. Such effects include ataxia, lethargy, painful extremities, peripheral neuropathies, convulsions, endocrine disorders, psychoses, nausea, vomiting, and abdominal pains (Bank, 1980). Chronic studies in rats exposed to thallium in their diet for 30 days exhibited marked growth depression and a nearly complete loss of hair (Clayton and Clayton, 1984). Histological changes noted for the skin of exposed animals included atrophic hair follicles and sebaceous glands.

Exposure to thallium salts during critical developmental stages in chicks and rats has been reported to be associated with the induction of adverse developmental outcomes (Karnofsky et al., 1950). Slight kidney changes were reported in pregnant rats exposed to thallium; fetal weight reduction in pups of exposed dams was also observed (Gibson and Becker, 1970).

Thallium has not been demonstrated to be carcinogenic in humans or experimental animals and may have some antitumor activity. In a study comparing the antitumor properties of several metal salts, thallium chloride was found to increase the median survival time to greater than seven times that of control animals; of the tumor-bearing rats, 25% were long-term survivors. No reports were found in the literature reviewed on the mutagenic activity of thallium or its salts.

EPA (1988a) developed an oral RfD for thallium of 7×10^{-4} mg/kg/day based on a study in which animals were exposed to increasing levels of thallium salts (i.e., thallous oxide, thallium acetate, thallium carbonate, thallium chloride, thallous nitrate, thallium selenite, and thallium sulfate) in the diet for 15 weeks. Alopecia and a slight increase in kidney weights were observed in the 15- and 30-ppm exposure groups; the 50-ppm group had 100% mortality by the 5th week of exposure. Using 5 ppm (0.39 mg/kg/day as

thallium) as the NOEL, and an uncertainty factor of 1,000 for an animal study with small group sizes, the reference dose of 7×10^{-4} mg/kg/day was derived.

Airborne concentrations determined to be protective of employee health and safety of 0.1 mg/m^3 have been promulgated by OSHA (1989).

TIN

Inorganic tin salts are only poorly absorbed following ingestion. The small amounts that are absorbed are distributed mainly to the bone, liver, and kidney. Inhaled tin tends to remain in the lungs.

The toxicity of inorganic tin salts depends on the particular compound involved. In animals, acute exposure to lethal doses results in diarrhea, internal bleeding, twitching, paralysis, and death (Barnes and Stoner, 1959). In humans, exposure to inorganic tin, eye and skin irritation has resulted (NIOSH, 1985b). Subchronic exposure to tin oxides, sulfide, and oleate in the diet produced no adverse effects in rats at dose levels up to 7900 ppm, while exposure to stannous chloride, orthophosphate, sulfate, oxalate, and tartrate at dose levels at or above 0.3% produced severe growth retardation, decreased food efficiency, anemia, and histological changes in the liver (deGroot et al., 1973). Chronic inhalation of fumes or dust containing tin oxides has been reported to cause stannosis, a benign pneumoconiosis in workers (Barnes and Stoner, 1959). In a careful study of 10 workers from a Chilean tin foundry, it was concluded that stannic oxide fumes were more likely than tin dust to cause stannosis. Persistent cough and phlegm were reported in males working in a tin smelter (Roach, 1966). In another study, no clinical symptoms were present and pulmonary X-rays of workers showed no evidence of fibrosis or silicosis (Robertson et al., 1961).

Stannous chloride was reported to be negative in long-term animal cancer bioassays in rats and mice following administration in both the drinking water and the diet (Kanisawa and Schroeder, 1967, 1969; NTP, 1982). No information

was available regarding the mutagenic or teratogenic properties of inorganic tin.

An oral RfD of 6.0×10^{-1} mg/kg/day has been promulgated by EPA (1988a), based on the National Toxicology Program (NTP, 1982) bioassay in which mice and rats orally exposed to stannous chloride developed nonneoplastic liver and kidney lesions (NTP 1982). An uncertainty factor of 100 was used to derive the RfD. OSHA (1989) has promulgated a time weighted average for tin of 0.1 mg/m^3.

VANADIUM

Vanadium, in most forms, is absorbed to a moderate extent. Short and long-term effects of vanadium exposure due to inhalation principally involve respiratory tract irritation, including coughing, wheezing, breathing difficulties, bronchitis, and chest pains. Eye irritation, skin irritation, and tongue discolorations have also been reported (NIOSH, 1977b; NAS, 1974). No data were found regarding the genotoxic potential or reproductive toxicity of vanadium. There is no evidence that vanadium has carcinogenic potential (NIOSH, 1977b).

An oral RfD of 5.7×10^{-3} mg/kg/day for ingestion exposure to vanadium (not otherwise specified) was derived by EPA (1987b). A time weighted average of 0.05 mg/m^3 has been set by OSHA as the workplace standard air concentration for vanadium.

ZINC

Zinc is absorbed in humans following oral exposure; however, insufficient data are available to evaluate absorption following inhalation exposure (EPA, 1984o). Zinc is an essential trace element necessary for normal health and metabolism and it is nontoxic in trace quantities (Hammond and Beliles, 1980). However, overexposure to zinc has been associated with a variety of adverse effects. Chronic and subchronic inhalation exposure to

zinc in humans has been associated with gastrointestinal disturbances, dermatitis, and metal fume fever, a condition characterized by fever, chills, coughing, dyspnea, and muscle pain (EPA, 1984o). Chronic oral exposure of humans to zinc may cause anemia and altered hematological parameters. Reduced body weights have been observed in rats administered zinc in the diet. There is no evidence that zinc is teratogenic or carcinogenic (EPA, 1984o).

EPA (1988) has derived an oral RfD of 2.1×10^{-1} mg/kg/day for zinc based on studies in which anemia and reduced blood copper were observed in humans exposed to oral zinc doses of approximately 2 mg/kg/day (Pories et al., 1967; Prasad et al., 1975). A safety factor of 10 was used in developing the RfD. For zinc oxide, a time weighted average was set by OSHA (1989). This value of 5 mg/m^3 has been determined to prevent any adverse effects in workers due to zinc oxide exposure. DHS has set applied action levels of 7.5 mg/l and 0.75 mg/m^3 for water and air concentrations of zinc, respectively (DHS, 1988c).

RADIONUCLIDES

Following inhalation or ingestion, radionuclides are absorbed into body tissues and irradiate nearby cells. The ultimate effects of the irradiation depend on the particular physical and chemical characteristics of the radionuclide (which determines its distribution and retention in the body) and its emitted radiation. For example, alpha particles have great ionizing power but because of their large size have poor penetrating power. Beta particles have less ionizing power but greater penetrating power. In humans, exposure to ionizing radiation can result in the induction of cancer, genetic disease, teratogenic effects, and degenerative changes. The target organs of the carcinogenic and degenerative effects are the respiratory tract, bone, liver, and the reticuloendothelial system (NRC, 1988).

EPA has not evaluated the carcinogenic potential for specific radionuclides. No cancer potency factors or RfDs are available for radioactive compounds.

4.0 HUMAN EXPOSURE ASSESSMENT

This section will address the potential pathways by which human populations could be exposed to contaminants at, or originating from, the NAS Alameda site study areas. In identifying potential pathways of exposure, both current and likely future land-use of the site and surrounding area will be considered.

An important step in identifying exposure pathways is to consider the mechanisms by which the chemicals of potential concern at the site may migrate in the environment. Fate and transport characteristics of the chemicals of concern are presented in Section 4.1. Migration pathways are discussed in Section 4.2 followed in Section 4.3 by a summary of the population and land use in the area near NAS Alameda. In Section 4.4, potential exposure pathways are presented. Section 4.5 evaluates the potential exposure pathways for each study area.

4.1 FATE AND TRANSPORT OF THE CONTAMINANTS OF CONCERN

Transport of chemicals in environmental media is a function of the physical and chemical properties of the chemical and of the environmental conditions at the site. The following section presents a general discussion of the chemical properties affecting mobility and chemical transformation and summarizes transport processes most likely to affect the chemicals detected at the NAS Alameda site.

Water solubility is a critical property affecting the environmental transport of a chemical: highly soluble chemicals can be rapidly leached from soils or waste and are generally mobile in groundwater. For inorganic contaminants, the solubility will depend on the valence state of the element and on the chemistry of the surrounding medium.

A compound's volatilization rate depends on its vapor pressure and water solubility. Highly water-soluble compounds generally have lower

volatilization rates from water than compounds having a low water solubility. Vapor pressure, a measure of the volatility of chemicals in their pure state, ranges from approximately 7×10^{-9} to 7.6×10^{2} mm Hg for liquids (EPA, 1986f). The Henry's Law constant, which is the ratio of a compound's vapor pressure (in atmospheres) to its solubility (in moles/m^3), is a more accurate measure of volatilization behavior than is vapor pressure for estimating releases to air from water. Compounds with Henry's Law constants greater than approximately 10^{-3} can be expected to volatilize readily from water. Those with values ranging from 10^{-3} to 10^{-5} volatilize less readily, while compounds with values less than 10^{-5} volatilize slowly (Lyman et al., 1982).

The octanol-water partition coefficient (K_{ow}) is often used to estimate the extent to which an organic chemical will partition from water into lipophilic tissues of organisms, such as fish or animal fat. The normal range of K_{ow} values is from -2.5 to 10.5. Chemicals with K_{ow} less than 3 are generally considered not to concentrate in animal tissues. Another measure of chemical uptake in animal tissue is known as the bioconcentration factor (BCF), which indicates the ability of a chemical to concentrate in biologic tissue. Similarly, the organic carbon partition coefficient (K_{oc}) reflects the propensity of a compound to adsorb to the organic matter found in soil. The normal range of K_{oc} values for all organic chemicals is from 10^{0} to 10^{7} (log K_{oc} = 1 to 7) with higher values indicating greater adsorption potential. Values of log K_{oc} less than 3 generally do not adsorb strongly enough to soils to affect overall leachability at normal soil organic content levels.

For inorganic contaminants, prediction of adsorption behavior is complex; the extent of adsorption depends on the soil content of organic matter, clay, and iron and aluminum hydroxides, as well as the pH of the groundwater. The affinity of a chemical for soil particles is defined as the soil partition coefficient, K_d, and is equated to the ratio of the concentration of the chemical on the soil to the concentration in the associated interstitial water. A value of 100 or greater is indicative of strong adsorption. Another indicator of reactivity of inorganic contaminants

is the redox potential (Eh) which is a measure of the ability to transfer electrons in solution.

The organic chemicals of potential concern can be classified into categories according to their similarity in chemical structure or physical/chemical properties (i.e., factors that would influence mobility in the environment). The chemical categories and the chemicals of concern within each category are shown in Table 4-1.

4.1.1 Organic Chemicals of Potential Concern

Monocyclic Aromatic Hydrocarbons

The monocyclic aromatic compounds detected at the NAS Alameda site, such as benzene, chlorobenzene, ethylbenzene, toluene, and xylene have high vapor pressures and relatively low water solubilities (Verschueren, 1983). The primary fate of these chemicals in surface soils or surface water is volatilization to the atmosphere (EPA, 1979), where they are rapidly destroyed by photooxidation (EPA, 1979). Monocyclic aromatics are moderately adsorbed by soils, and their transport is generally retarded relative to groundwater. The chemicals have a liquid density less than water and may form a separate phase above the water table if present in sufficient quantity (e.g., as part of a gasoline plume). Vapor-phase diffusion may be a significant transport process in unsaturated soils.

The predominant fate processes affecting monocyclic aromatics are sorption to soils, volatilization with subsequent photochemical oxidation (EPA, 1979), and biological transformation in some soils (Barker et al., 1987). Transformation byproducts of alkyl aromatics are cresols and carbon dioxide (Barker et al., 1987, Gibson et al., 1966). Monocyclic aromatics are not appreciably concentrated in plant or animal tissues (EPA, 1979).

Table 4-1

PHYSICAL AND CHEMICAL PROPERTIES OF THE ORGANIC CHEMICALS OF POTENTIAL CONCERN

	Molecular Weight (g/mole)	Water Solubility @20°C (mg/l)	Vapor Pressure @ 20-25°C (mm Hg)	Henry's Law Constant (atm-m3/mole)	Octanol: Water Partition Coefficient (log K_{OW})	Organic Carbon Partition Coefficient (log K_{OC})
Monocyclic aromatic hydrocarbons						
Benzene	78.0	1787	95	5.4×10^{-3}	2.12	1.92
Chlorobenzene	112.6	466	12	3.7×10^{-3}	2.84	2.52
Ethyl benzene	106	152	7	6.4×10^{-3}	3.15	3.04
Toluene	92.2	515	22	6.6×10^{-3}	2.69	2.48
Xylenes (mixed)	106.2	200	6	7.0×10^{-3}	2.95	2.38
Chlorinated aliphatic hydrocarbons						
1,2-dichloroethylene (cis/trans)	96.9	600-800	208	6.6×10^{-3}	0.48	1.77
carbon tetrachloride	153.8	760	90	2.4×10^{-2}	2.64	2.04
methyl chloroform (1,1,1-TCA)	133.4	1,500	123	1.4×10^{-2}	2.50	2.18
methylene chloride	84.9	20,000	349	2.0×10^{-3}	1.30	0.92
trichloroethylene	130.9	1,100	58	8.9×10^{-3}	2.42	2.20
Organochlorine pesticides and byproducts						
aldrin	364.9	0.027	7.5×10^{-5}	2.7×10^{-5}	5.30	4.98
chlordane	409.8	0.006	2.5×10^{-5}	1.9×10^{-3}	4.78	4.00
p-p' DDT	354.5	0.0055	5.5×10^{-7}	5.0×10^{-5}	6.19	5.38
Endosulfan (alpha)	406.95	0.53	1×10^{-5}	1×10^{-6}	3.55	3.98
Endrin	380.9	0.20	2.7×10^{-7}	1×10^{-7}	5.34	4.03
Endrin aldehyde	380.9	0.25	2×10^{-7}	4×10^{-7}	4.70	NA

NA: value not available (a) see Table 4-2

Sources: MacKay and Shiu 1981, Verschueren 1983, Weast 1983

Table 4-1 (Continued)
PHYSICAL AND CHEMICAL PROPERTIES OF THE ORGANIC CHEMICALS OF POTENTIAL CONCERN

	Molecular Weight (g/mole)	Water Solubility @20°C (mg/l)	Vapor Pressure @ 20-25°C (mm Hg)	Henry's Law Constant (atm-m3/mole)	Octanol: Water Partition Coefficient (log K_{OW})	Organic Carbon Partition Coefficient (log K_{OC})
Organochlorine pesticides and byproducts (cont'd)						
Heptachlor	373.3	0.056	3×10^{-4}	3×10^{-3}	4.40	3.78
Lindane	290.8	7.5	1.6×10^{-4}	3.1×10^{-6}	3.85	3.03
Herbicides						
2,4-D	221.0	620	4×10^{-1}	1.9×10^{-4}	2.81	1.30
Bromacil (Krovar I, Chlorvar)	261.1	815	8×10^{-4}	3.4×10^{-7}	NA	NA
Diuron (Krovar I, Telvar)	233.1	42	3×10^{-6} @30°	2.2×10^{-8}	2.77	NA
Monuron (Telvar)	198.6	262	5×10^{-7}	NA	NA	1.91
Glyphosate isopropylamine salt (Roundup)	228.2	12,000	NA	NA	NA	NA
Simazine (Princep)	201.7	3.5	6×10^{-9}	4×10^{-10}	NA	NA
Other pesticides						
Diazinon	304.4	40	4.1×10^{-4}	NA	NA	NA
Malathion	330.4	145	1.2×10^{-2}	3.7×10^{-7}	2.89	3.25
Warfarin	308.3	17	6.7×10^{-2}	1.6×10^{-3}	2.52	2.75
Ketones						
Acetone	58	1,000,000	270	2×10^{-5}	-0.24	0.34
Methyl ethyl ketone	72	353,000	77.5	2.7×10^{-5}	0.26	0.65

NA: value not available (a) see Table 4-2
Sources: MacKay and Shiu 1981, Verschueren 1983, Weast 1983

Table 4-1 (Continued)

PHYSICAL AND CHEMICAL PROPERTIES OF THE ORGANIC CHEMICALS OF POTENTIAL CONCERN

	Molecular Weight (g/mole)	Water Solubility @20°C (mg/l)	Vapor Pressure @ 20-25°C (mm Hg)	Henry's Law Constant (atm·m3/mole)	Octanol: Water Partition Coefficient (log K_{OW})	Organic Carbon Partition Coefficient (log K_{OC})
Polychlorinated biphenyls	189-399	0.003-0.6	10^{-5} - 10^{-3}	10^{-4} - 10^{-3}	4.7-6.8	NA
Polycyclic aromatic hydrocarbons	(a)	(a)	(a)	(a)	(a)	(a)
Phenols						
Phenol	94.1	93,000	3×10^{-1}	4.5×10^{-7}	1.46	1.15
2,4-Dimethyl phenol	122	590	2.6×10^{-2}	1.7×10^{-5}	2.81	1.96
Bis-2-ethylhexyl phthalate	391	0.4	2.0×10^{-7}	4.4×10^{-7}	5.11	4.94

NA: value not available (a) see Table 4-2

Sources: MacKay and Shiu 1981, Verschueren 1983, Weast 1983

Phenols

This class of compounds is highly water soluble and mobile in soils and groundwater; phenol is more readily transported than alkyl phenols. Phenol is a weak acid, which is found almost entirely in the neutral (non-ionized) form in natural waters. Photooxidation may be an important fate process of phenol in aerated, clear surface waters (EPA, 1979). Phenol may also be nonphotolytically oxidized by suspended iron or copper in aerated waters. Microbial degradation of phenols in soils and surface waters may occur when adequate populations of microorganisms are present. Typical degradation products include catechol and 2-hydroxymuconic semialdehyde (EPA, 1979). Phenol can also undergo condensation reactions with other phenolic compounds to form polymeric complexes. Bioconcentration does not appear to be an important fate process for phenol.

Chlorinated Aliphatic Hydrocarbons

The chlorinated aliphatic hydrocarbons are volatile, moderately to poorly water-soluble compounds. All the chemicals in this group are liquids at room temperature except vinyl chloride, which is a gas at room temperature. The densities of these chemicals are greater than water. Therefore, they may form a separate phase at the bottom of an aquifer if present in sufficient volume. Chlorinated aliphatic hydrocarbons tend to be poorly adsorbed to soils and to persist in groundwater and subsurface soils. Tetrachloroethylene, trichloroethylene, 1,1,1-trichloroethane (methyl chloroform) and cis- and trans-1,2-dichloroethylene have been shown to be slowly transformed to vinyl chloride in anaerobic sediments (Barrio-Lage et al., 1986; Vogel et al., 1987; Wilson et al., 1986). The rate and extent of these reactions are highly dependent on site-specific factors such as nutrient availability and microbial composition of the soil. The ultimate fate of chlorinated aliphatic hydrocarbons in surface soils and surface water is generally volatilization to the atmosphere and subsequent photooxidation (EPA, 1979). These compounds are not significantly bioaccumulated.

Ketones

The ketones detected at NAS Alameda, acetone and methyl ethyl ketone (MEK or 2-butanone), are miscible in water, and therefore have low Henry's Law constants and do not volatilize readily from water or wet soil. The ketones are poorly adsorbed by soils and highly mobile in groundwater. Ketones are rapidly biotransformed in soils and natural waters and eventually degrade to carbon dioxide. Based on their low K_{ow}s, acetone and MEK would not be expected to bioaccumulate in plant or animal tissue.

Organochlorine insecticides

The organochlorine insecticides of potential concern are poorly soluble, relatively nonvolatile organics which are moderately to strongly adsorbed to soils. The most important fate and transport processes affecting each of the pesticides are discussed below:

- ALDRIN: Based on its low water solubility and high K_{ow}, aldrin will not leach extensively from soils with high organic content, but low levels could appear in groundwater. Aldrin has been reported to volatilize from natural waters with a half-life of days (EPA, 1979). Aldrin is biotransformed in water to dieldrin and may also undergo phototransformation in water to the compounds photoaldrin and photodieldrin. Aldrin has been reported to bioaccumulate in aquatic organisms.

- CHLORDANE: Technical chlordane consists of a liquid mixture of more than 45 chlorinated compounds, which include two isomers of the chlordane molecule, heptachlor, chlordene, and other constituents in varying proportions (EPA, 1979). The following discussion relates to the two chlordane isomers, which constitute approximately 40 percent of the technical chlordane mixture. The most important fate processes affecting chlordane are sorption to soils or sediments, and photolysis in surface waters. Based on its log K_{ow}, chlordane would be strongly adsorbed to soils and sediments, and would not be mobile in groundwater. Chlordane is highly persistent in soils and is generally resistant to biodegradation. Under exposure to light, chlordane has been reported to convert to an isomer with higher toxicity and greater bioaccumulation potential (Haque, 1970). Chlordane is strongly bioaccumulated in fish (BCF=3,600 - 19,000) and has been found to concentrate in animal fats and milk (EPA, 1979).

- DDT: This pesticide consists of a mixture of three isomers (p,p', o,p', and o,o') in an approximate ratio of 7:3:1. The isomers have similar physical/chemical properties and may be considered as one chemical for the purpose of this discussion. The dominant fate and transport characteristics of DDT are its sorption to solid materials, persistence in environmental media, and volatilization. As indicated by its high K_{ow}, DDT will preferentially sorb to sediments in aquatic systems, and will be strongly bound to soils with a high organic carbon content. DDT on surface soils is likely to be transported along with soil particles by runoff or wind. In spite of its low Henry's constant, volatilization is an important process leading to eventual loss of DDT from surface waters, since the pesticide is generally not significantly degraded by chemical or biological mechanisms. Reported half-lives for biotransformation of DDT in soils range from 2 years to >15 years (HSDB, 1988). Major products of biodegradation include DDD and DDE. DDT is strongly bioaccumulated in both aquatic and terrestrial organisms (BCFs range from 10^3 - 10^5), and is also concentrated through the food chain (HSDB, 1988).

- ENDOSULFAN: The primary processes affecting endosulfan in the environment are sorption to soils or sediments, oxidation and biotransformation to endosulfan sulfate, and possibly volatilization from surface waters (EPA, 1979). Endosulfan may be taken up by fish, but is also readily excreted; bioaccumulation is therefore not likely to be an important process (EPA, 1979).

- ENDRIN: Endrin is very insoluble in water and has a low vapor pressure. Its low Henry's constant indicates that endrin will not volatilize rapidly from surface water. This pesticide would be strongly adsorbed to soils and would not migrate extensively to groundwater. Microbial cultures from soils were reported to degrade endrin; the primary metabolite was endrin ketone (EPA, 1979). Endrin is bioaccumulated in aquatic organisms; bioconcentration factors ranging from 10^3 to 10^4 have been reported for various species. Endrin aldehyde occurs as an impurity in endrin, and is also a metabolite of endrin (EPA, 1979). The primary fate of this chemical in the environment is generally sorption to soils and sediments, and transport along with soil particles via wind or overland runoff. Volatilization is not expected to be a significant fate for endrin aldehyde. Based on its K_{ow}, endrin aldehyde may be concentrated by aquatic organisms.

- HEPTACHLOR: This pesticide is a component of technical chlordane. Based on its K_{ow}, heptachlor would be moderately adsorbed to soils with a high organic content. Volatilization from surface soils and surface waters is an important pathway of loss from these media. The half-life of heptachlor in soils was reported to be 6 months (EPA, 1985aa). Heptachlor is transformed by some soil organisms; transformation products include 1-hydroxychlordene and heptachlor epoxide (EPA, 1979). Heptachlor is bioconcentrated in aquatic and terrestrial organisms; reported bioconcentration factors range from 10^4 to 10^5 (EPA, 1979).

- BENZENE HEXACHLORIDE (GAMMA-BHC OR LINDANE): This pesticide has a low vapor pressure and a low solubility in water. Lindane is moderately adsorbed to sediments and soils, but will leach slowly from soils to groundwater (HSDB, 1988). The primary fate of lindane in aquatic systems is biotransformation in sediments. Disappearance half-lives on the order of several days to a year have been reported (EPA, 1979) for lake and estuary systems. Biodegradation is more rapid in anaerobic soils and sediments than in aerobic soils and sediments. Products of lindane biotransformation include other isomers of BHC, tetrachlorobenzenes, and chlorinated cyclohexanes (EPA, 1979). Lindane is slightly to moderately bioaccumulated in aquatic organisms: fish BCFs of 200 to 1470 have been reported.

Herbicides

- 2,4-D (2,4-DICHLOROPHENOXYACETIC ACID): This herbicide is highly soluble in water and may be leached readily from some soils. 2,4-D is reported to be hydrolyzed in soil to acetic acid and dichlorophenol, which in turn are transformed to non-toxic byproducts (White-Stevens, 1976, as cited in HSDB 1988). The disappearance rate in surface soils is on the order of days to weeks. 2,4-D is not strongly bioconcentrated in aquatic organisms (BCF = 4.9 - 150) (Kanazawa, 1978).

- BROMACIL (KROVAR I, CHLORVAR): Krovar I is a powdered mixture of bromacil and diuron, which may be applied dry or in water. Bromacil is moderately water soluble and will leach readily from soils to groundwater. Volatilization and biodegradation losses from soils are likely to be minor (HSDB, 1988). Bromacil was reported to be highly resistant to photolysis in soils (Verschueren, 1983).

- DIURON (KROVAR I, TELVAR): After application to surface soils, Diuron is reported to remain in the top 5-10 cm of soil, with a half-life of approximately one year (Jury et al., 1984). Transformation has also been reported in anaerobic soils and in surface waters and sediments (HSDB, 1988).

- MONURON (TELVAR): This herbicide is a solid at room temperature. The chemical has low water solubility and low vapor pressure. Monuron is moderately to strongly absorbed to soils; leaching may occur with high rainfall or low soil organic content (EPA, 1975). The primary mechanism leading to loss from soils is generally biological transformation; the reported half-life in soils ranges from 1.5 to 6 months (Haque and Freed, 1975).

- SIMAZINE (PRINCEP): This triazine herbicide is strongly adsorbed to soils; however, trace amounts of the chemical may leach to groundwater. The primary processes affecting simazine in soils are transport with soil particles and biodegradation (reported half-life was 17 to 52 days). No information was available on persistence in other media (HSDB, 1988).

- GLYPHOSATE ISOPROPYLAMINE SALT (ROUNDUP, GLYPHOSATE): Little information is available on the chemical properties of this herbicide, or the processes it undergoes in the environment. Roundup is the isopropyl amine salt of an organic acid, and is therefore highly water soluble. It is generally sold as an aqueous solution (HDSB, 1988).

Other Pesticides

- DIAZINON: The most important environmental processes affecting this organophosphate pesticide are volatilization from surface water and soil and transformation by biotic and abiotic mechanisms. Disappearance times on the order of days to weeks have been reported for diazinon applied to surface soils (Spencer, 1982; Menzie, 1974). Bioconcentration factors of 5 to 150 were reported for fish and other aquatic organisms (Kanazawa, 1978).

- MALATHION: In its pure form this pesticide is a liquid. It is moderately adsorbed to soils and sediments, and will not be leached extensively to groundwater. Malathion has a low vapor pressure, and volatilization from soil or surface water will not be an important process. Biodegradation and hydrolysis may be important fates for this chemical in both surface water and soil. Disappearance rates of 10 and 25 days were reported for soil and estuarine water, respectively (Walker et al., 1973). The degradation rate in soil was shown to be highly dependent on moisture content and pH. Malathion was reportedly not bioconcentrated in freshwater fish, but moderate uptake by shrimp was reported (BCF=150 - 1917) (Conte and Parker, 1975; Walsh and Ribelin, 1973).

- WARFARIN: This rodenticide is generally used in dust or pellet form, and generally is not released to the environment in large quantities. Therefore, little information exists on its environmental fate. Based on its chemical properties, warfarin would be moderately adsorbed to soils; volatilization may be a significant transport pathway. Once in the atmosphere, warfarin would be rapidly destroyed by photooxidation (Gore, et al., 1971).

Other Organics

PHTHALATES

Phthalates are ubiquitous in the environment due to their use as plasticizers and are also commonly observed in field and laboratory blanks. Bis(2-ethylhexyl)phthalate is strongly absorbed to soils and sediments, and would not be expected to be significantly transported in groundwater (Russell and McDuffie, 1986). Phthalates have low volatilities, and would tend to be transported along with soil particles in air or runoff.

POLYCHLORINATED BIPHENYLS (PCBs)

The chemical, physical and biological properties of polychlorinated biphenyls (PCBs) vary widely, depending on the number and location of the chlorine atoms on the PCB molecule. As a class of compounds, PCBs are relatively inert, and therefore persistent in the environment. In general, PCBs have low water solubilities and low vapor pressures. Volatilization of PCBs from surface waters and surface soils may be an important transport process for PCBs with fewer than four chlorines, as indicated by their relatively high Henry's Law constant (10^{-3} atm-m^3/mol). The loss rate from surface waters is expected to be highly dependent on site-specific factors such as temperature, wind speed, and the concentration of dissolved and suspended organic matter in the water.

Once transported to the atmosphere, PCBs may undergo photooxidation. Atmospheric half-lives of specific PCB compounds range from 12.9 days to 1.31 years, and generally increase with increased chlorination (EPA, 1987o). Atmospheric PCBs exist primarily in the vapor phase; removal of PCBs from the atmosphere and return to earth in precipitation is an important dispersion mechanism for these compounds (Eisenreich et al., 1981).

Their low water solubility and high octanol-water partition coefficients indicate that PCBs have a strong affinity to adsorb to organic materials in soils and sediments. Experiments have confirmed the preferential partitioning of PCBs to soil or sediments in a soil-water system (EPA, 1979). Transport in surface runoff of PCBs adsorbed to suspended particles and sediments may be an important transport mechanism. Leaching of PCBs from soil into groundwater is not anticipated under most conditions. However, organic solvents or oil will mobilize PCBs in soils (Griffin and Chou, 1981), and PCBs may travel through the subsurface as a separate phase if present in large amounts.

Biodegradation is the only transformation process known to be significant for PCBs in soils and sediments (ATSDR, 1988b). The relative

rates of biological transformation depend on the position and number of chlorine atoms. PCBs with one to three chlorines biodegrade relatively quickly; PCBs with four chlorines biodegrade slowly; and PCBs with five or more chlorines are resistent to biodegradation. In addition to degree of chlorination, chlorine position on the biphenyl molecule is important in determining the biodegradation rate. Experimentally determined bioaccumulation factors for PCBs in aquatic organisms range from 20,000 to 660,000 (Leifer et al., 1983).

POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)

The term polycyclic aromatic hydrocarbons describes a diverse class of chemicals consisting of 2 or more fused benzene rings, which vary widely in arrangement. PAHs are chiefly formed from the combustion of organic material and are widely distributed in the environment due to both natural and anthropogenic activities. Major sources of PAHs in the environment include petroleum derivatives such as creosote, asphalt, and fuel oil, and combustion of wood or coal.

The physical properties of PAHs, which in turn affect their mobility in the environment, depend largely on the sizes of the molecules; therefore, molecular weights are key properties. Table 4-2 lists physicochemical properties of the priority pollutant PAHs. Vapor pressures and solubilities vary widely within the class and can be correlated with molecular weight. Volatilization may be a significant transport mechanism for a low molecular weight PAH such as naphthalene, but much less significant for the higher molecular weight compounds, such as indeno(1,2,3-c,d)pyrene. The relatively high solubilities of the lower molecular weight PAHs can result in some migration into groundwater. The higher molecular weight PAHs tend to adsorb strongly onto soil surfaces, especially if the soil has a high organic carbon content. For this reason, overland transport of PAHs adsorbed onto soil particles is a typical mechanism of migration. PAHs associated with fuel oil may also migrate through the unsaturated zone or through an aquifer as a non-aqueous plume.

Table 4-2

Selected Physical and Chemical Properties of Some Polycyclic Aromatic Hydrocarbons

Compound	Molecular Weight	Vapor Pressure (mm Hg) (20-25 °C)	Water Solubility (mg/liter)	Log K_{ow} [a]	BCF [b]
Naphthalene	128	0.049-0.078	31.7-34.4	3.37	146
Phenanthrene	178	1.2×10^{-4}	1.3	4.46	1,230
Anthracene	178	6.0×10^{-6}	0.070	4.45	1,210
Flouranthene	202	9.2×10^{-6}	0.260	4.90	2,920
Pyrene	202	4.5×10^{-6}	0.140	4.88	2,800
Benz[a]anthracene	228	2.1×10^{-7}	0.014	5.61	11,700
Chrysene	228	6.4×10^{-9}	0.002	5.61	11,700
Benzo[a]pyrene	252	5.5×10^{-9}	0.00025-0.0038	6.06	28,200
Benzo[g,h,i]perylene	276	1.0×10^{-10}	0.00026	6.51	68,200

Sources: EPA (1979); Radding et al. (1976); Verscheuren (1983).

[a] K_{ow} = octanol/water partition coefficient

[b] BCF = bioconcentration factor, values estimated from the equation of Veith et al. 1979

PAHs are capable of undergoing photolytic transformations in the environment, and their degradation half-lives vary widely. Photolysis is a process that is greatly affected by site-specific variables such as intensity of sunlight, turbidity of water (if in a surface water environment), depth in surface water, or nature of the adsorbent.

Biodegradation of PAHs in the environment is extremely variable across the chemical class. The process is highly complex and depends on numerous factors such as the species of microorganisms present, availability of nutrients, oxygen tension, degree of acclimation, nature of the medium, concentration of the chemical, temperature, and pH. In general, the di- and tricyclic PAHs are more readily biodegraded than the tetracyclic and higher polycyclic hydrocarbons (Tabak et al., 1981).

4.1.2 Inorganic Chemicals of Potential Concern

ANTIMONY

Antimony (Sb) is a metallic element occurring in the earth's crust in concentrations of approximately 0.2 to 0.5 mg/kg (Arthur D. Little, 1976). It can exist in four oxidation states: +5, +3, 0, and -3. The +5 form is important only in highly oxidizing environments. The chemistry of antimony is similar to that of arsenic, which directly overlies it in the periodic table. Antimony forms trivalent and hexavalent compounds with sulfur and chloride.

Under moderately oxidizing aqueous conditions, antimony occurs primarily in the +3 oxidation state, as the hydrated trioxide $Sb_2O_3(H_2O)_n$. Salts of antimonite acid can also be formed in natural waters. The oxides and salts of antimony are moderately soluble in water; therefore, antimony is considered to be mobile in surface waters (Cotton and Wilkinson, 1982). Under conditions of high acidity and in contact with reducing agents, the poisonous gas stibine (SbH_3) may be formed. Stibine is volatile and soluble in water (5000 mg/L), but is rapidly oxidized to soluble oxides under aerobic conditions (Parris and

Brinkman, 1976). Volatilization may be of limited importance in loss of antimony from surface or groundwater.

The sorptive behavior of antimony has not been well characterized; however, as with arsenic, sorption is expected to be a major factor limiting aqueous transport. Crecelius et al. (1975) reported that antimony in the marine environment may coprecipitate with iron, manganese, or aluminum oxides, and could then be available for remobilization under reducing conditions. Very little antimony was found to be complexed with humic acids. Antimony was shown to persist in soils (Crecelius et al., 1974) and in river sediments (Maxfield et al., 1974). Baes et al. (1984) reported a soil-water distribution coefficient (K_d) of 45 for antimony.

Bioaccumulation and biotransformation of antimony compounds may be of importance in some environments. Based on analogy to arsenic, biological formation of trimethyl stibine would be expected to occur; however, this process has not been detected in environmental samples (EPA, 1979). Bioconcentration of antimony has been shown to occur in fish (BCF = 40), and invertebrates (BCF = 16000) (EPA, 1979).

ARSENIC

Arsenic (As) has four stable oxidation states, +5, +3, 0, and -3. Arsenates (As(V)) predominate in most soils, while arsenites (As(III)) may dominate in reducing environments such as flooded soils. As(III) species are generally more mobile than As(V) in subsurface soils. There is evidence that arsenic may leach into groundwater, especially from soils with low sorptive capacity (EPA, 1984q). The primary processes limiting the mobility of arsenic in soils are precipitation as metal salts and adsorption to amorphous metal oxides. In highly reduced soils, or in the presence of certain microorganisms and fungi, arsenic may be reduced to arsine (-3). Arsines may be metabolized by soil microorganisms to trimethyl arsine, a volatile and highly toxic compound. Loss of arsenic by volatilization has been observed in a number of soils (Cox, 1975).

As(V) and As(III) are the most common forms in aqueous environments. The two states are readily interconverted by biological and physicochemical redox reactions. The fate of arsenic in aquatic media is influenced by numerous factors (EPA, 1984q). Arsenic is strongly adsorbed by amorphous metal oxides, and may also be adsorbed by organic matter or clay. Bioconcentration factors for arsenic in aquatic organisms range from 333 to 6,000 (EPA, 1984q).

Estimation of arsenic's partitioning coefficient, K_d, is complicated by the dependence on redox conditions and pH. Baes et al. (1984) reported a theoretical K_d of 200 mL/g. Baes and Sharp, (1983), however, reported a range of 0.1 to 8.3 mL/g for As(III); other investigators reported K_ds in the range of 1.9 to 18 mL/g (Frost and Griffin, 1977). In work with sewage sludge, Gerritse et al. (1982) reported ranges of about 15-30 mL/g for sandy loam soil and 5 - 10 mL/g for sandy soil. These experimental K_ds, which are much lower than the value given by Baes et al. (1984), may more accurately reflect the mobility of arsenic in soils.

ASBESTOS

It has generally been thought that the mineral fibers grouped under the term asbestos are not subject to significant chemical or biological transformation in the environment (EPA, 1979). However, recent evidence suggests that the chrysotile species undergoes variable amounts of environmental/biological transformation (Seshan, 1983). It has been reported that chrysotile will react with weak acids and water, causing magnesium (and some silicon) to be released from the crystalline structure. Thus, to a limited extent, degradation of chrysotile may occur through acid-water interactions in the environment.

Asbestos does not absorb onto soil and sediment particulates. However, certain species of trace minerals and organic compounds do have a finite affinity for asbestos and will form a suspension that ultimately settles to

the sediment layer. This affinity is due to either chemical reactivities between surface groups or to electrostatic attraction based on the surface charge of asbestos fibers. Thus, because of the leaching of magnesium from chrysotile fibers in aqueous solutions, the surface charge of this form of asbestos is continuously changing (Seshan, 1983). These changes may increase the potential for asbestos degradation in the environment.

All of the available evidence indicate that aquatic species do not bioaccumulate asbestos fibers. There is a possibility that the leaching of metal ions from ingested chrysotile asbestos may result in the accumulation of heavy metals in some aquatic organisms. The environmental significance of these processes is not clear at this time.

Because most asbestos is highly resistant to thermal and chemical degradation, it persists in the environment and can be widely distributed by both natural forces and human activities. The magnitude of this redistribution depends on a myriad of factors including: rates of air and water flow, fiber diameter, rainfall, thermal air conversions, agglomeration of particles, and human activities that may disturb settled asbestos (e.g., activity at an uncapped asbestos-containing landfill). There is little information available on the ultimate fate of asbestos fibers in the environment. While it is reported that fibers are readily subdivided by mechanical means into smaller fibrils (<1.0 um), it is not known if fibers are subdivided by natural events.

Dispersion of asbestos emitted to the atmosphere is governed by fiber length, topography, meteorological conditions, and the emission source. Wind speed and temperature stratification are important factors. Fibers moving through the atmosphere tend to fall to the ground as a result of gravity and rain. Asbestos emitted to air in the form of "large" agglomerated particles will settle to earth relatively quickly and have a limited potential for airborne contamination. Studies of atmospheric migration of asbestos originating from mines and mills have indicated that asbestos dust may be transported as far as 27 kilometers from the source (EPA, 1986h).

Asbestos can travel considerable distances in water, primarily the result of the charged nature of the asbestos fibers as discussed above. Suspensions of asbestos particles will remain in water until the various degradation processes result in its eventual sedimentation. Particles have been reported to move several hundred miles or more in Lake Superior. High river flows in areas surrounding Philadelphia and Atlanta have resulted in high fiber counts in the city water supplies (Millette et al., 1983).

BARIUM

Barium (Ba) can form compounds with acetate, nitrate, chloride, and hydroxide, but these compounds are very soluble. In soils containing these compounds, barium's mobility is increased (EPA, 1985h). Barium is likely to stay in solution in the absence of adsorptive carbonate or clays (EPA, 1984i). Barium has not been found to form complex ions in water (Cotton and Wilkinson, 1982).

BERYLLIUM

Beryllium (Be) is found predominantly in the +2 oxidation state. Elemental beryllium is found only under strongly reducing conditions. The element tends to form very stable, covalent compounds with ions such as fluoride and hydroxide (Cotton and Wilkinson, 1982). Beryllium hydroxide ($Be(OH)_2$) is formed when soluble beryllium salts are dissolved in water. The hydroxide is not very soluble in the pH range of most natural waters (pH 4-8), and the major species contributing to beryllium solubility is the hydrated ion (Pourbaix, 1963).

Beryllium is not a well-studied element, but it is chemically similar to aluminum, so that parallels between the two elements can be drawn to predict beryllium's fate. Thus sorption to clay mineral surfaces is expected at low pH. Beryllium is expected to sorb to clay particles in neutral to moderately

acidic surface soils. If beryllium reaches the groundwater, the relatively insoluble hydroxide should form.

In a study of eleven surface soils, beryllium had a low to moderate mobility, which was related to the clay content, surface area, iron oxide concentration, and pH (Korte et al., 1976).

CADMIUM

Cadmium (Cd) is generally found in the +2 oxidation state in the environment (Cotton and Wilkinson, 1982). The pH of the environment in which it is found will determine the speciation of cadmium through the formation of hydroxides: $Cd(OH)^{+}$, $Cd(OH)_2$, $Cd(OH)_3^{-1}$, and $Cd(OH)_4^{-2}$. Under reducing conditions and in the presence of sulfur, the relatively insoluble cadmium sulfide (CdS) will form. Therefore, under anaerobic conditions, CdS will be expected to control cadmium solubility (EPA, 1979).

Cadmium appears to move slowly through soil, and its mobility is dependent on adsorption-desorption processes (Kabata-Pendias and Pendias, 1984). Adsorption is influenced by the clay (Korte et al., 1976) and metal oxide (EPA, 1979) content of the soil and sediments. Adsorption is also a pH-dependent process that increases with increasing pH. Below pH 6 to 7, desorption is the dominant process. Huang et al. (1977) found that organic anions enhance adsorption even at low pH levels. The organic acids react with cadmium to form an organo-cadmium complex, which can then be adsorbed to soil or sediment particles.

Organic materials such as humic and fulvic acids can influence the speciation of cadmium. Gardiner (1974) found that the amount of Cd^{+2} in lake water depends on the pH and the amount of organic matter present. A cadmium-organic complex was found to bind cadmium at pH levels as low as 3 (Guy and Chakrabarti, 1976). O'Shea and Mancy (1978) reported that an increase in hardness resulted in a decrease in complex formation.

CHROMIUM

Chromium (Cr) can be found in oxidation states ranging from -2 to +6. Cr(III) is the most commonly occurring oxidation state, and Cr(VI) is the next most frequently encountered. Cr(V) and Cr(IV) are unstable intermediates that occur during the reduction of Cr(VI) to Cr (III). The lower oxidation states are only formed under strongly reducing conditions, and of these oxidation states, only Cr(II) is found in aqueous solutions (Cotton and Wilkinson, 1982; Moore and Ramamoorthy, 1984).

Cr(III) is the predominant form under the pH and redox conditions commonly found in natural waters and soils. Cr(III) tends to form stable complexes with both organic and inorganic anions, and those anions should remove Cr(III) from solution. Most of the Cr(III) found in soils is in mixed Cr(III) and Fe(III) oxides or in the lattice of minerals, although Cr(III) complexed with organic ligands may stay in solution for over a year (James and Bartlett, 1983). Cr(III) is mobilized only in a very acidic soil media. The adsorption of chromium onto clays is pH-dependent.

COBALT

Cobalt (Co) generally occurs in the 0 or +2 oxidation states. Elemental cobalt is relatively unreactive, and is quite stable in air or water. Cobalt appears to occur infrequently in soluble form in natural aquatic systems. Several surveys show that cobalt is generally not detected in water and that concentrations greater than 10 μg/liter are rare (NAS, 1977). The most important control on the mobility of cobalt in aquatic and terrestrial systems is probably adsorption to clay minerals and to the hydrous oxides of iron, manganese, and aluminum. Small amounts of cobalt may be solubilized by bacteriological activity. Cobalt is an essential element and can be accumulated by plants and animals, through generally not to excessive concentrations (NAS, 1977). Atmospheric transport of cobalt and cobalt compounds can occur.

COPPER

Copper (Cu) can be found in three oxidation states: 0, +1 and +2. Of these three, only the Cu(II) oxidation state is found in aquatic systems. In polluted environments, copper can also form complexes with cyanide, amino acids, and humic substances. In the absence of organic complexing agents, hydrolysis and precipitation dominate copper's chemistry in aqueous environments.

The interactions of copper with organic materials in natural waters have been studied extensively. Organo-copper interactions result in the increased solubility of some copper-containing minerals and the subsequent transport of the organocupric complex (Ong et al., 1970; Rashid and Leonard, 1973). Hydrous metal oxides can sorb copper and render it immobile (Jenne, 1968). This sorption process occurs in competition with binding of other metals, and competitive adsorption could result in the release of copper. Copper is not very mobile in sediments. Adsorption, precipitation, and organic complexation are also important processes in soils (Kabata-Pendias and Pendias, 1984). These processes render copper one of the least mobile metals.

CYANIDES

The behavior of cyanide (CN) compounds in soils has not been widely studied. The fate and transport of cyanides will depend upon their form. The simplest cyanide, hydrogen cyanide (HCN), is an acid and can dissociate into the cyanide ion (CN^-). HCN and CN^- are jointly referred to as free cyanide.

The simple cyanides are comprised of HCN and those salts formed with Group I and Group II metals, such as NaCN and $Ca(CN)_2$. These rapidly dissociate in water, leaving CN^- to hydrolyze or to react with trace metals. Iron, gold, cadmium, copper, nickel, silver, and zinc have also been found to form simple metallocyanides (Fuller, 1977; 1978). Simple metallocyanides have solubilities ranging from essentially insoluble (copper and nickel) to highly soluble (barium and potassium).

IRON

Iron (Fe) is a relatively immobile element. Biological and chemical reactions cause precipitation of iron in soils, which accounts for its immobility. Fe(II) is more soluble than the Fe(III) form, and hence, more mobile in soils, though it was found that Fe(II) from landfill leachate migrated only slowly under highly acidic conditions (Fuller, 1978). In aquatic media, iron can undergo many different chemical reactions which depend on pH and oxidation-reduction potential. It was found that greater than 82 percent of dissolved iron is chelated with dissolved organic carbon ligands (Steinburg, 1980).

LEAD

Lead (Pb) is found in the +2 and +4 oxidation states. The most commonly found ion in the environment is Pb^{+2}. Metallic lead can be formed under reducing or basic conditions when very little sulfur is present (Cotton and Wilkinson, 1982). The predominant fate of lead in the environment is sorption to soils and sediments. Carey et al. (1980) found lead concentrated in the surface soils. The adsorption of lead is pH dependent, increasing with increasing pH. Above pH 7, essentially all lead in soil is sorbed (Huang et al., 1977). Korte et al. (1976) found that lead was virtually immobile in all but sandy soils.

There is some disagreement about the precise distribution of lead in aquatic systems (EPA, 1979). However, it is generally agreed that lead concentrations in water decrease over time and that sorption of lead to both sediments and dissolved particulates is the favored process, with clay, hydrous metal oxides, and organic matter influencing this process. The presence of the clays and oxides will enhance the sorption of lead to the soils. Organic matter will react with the lead to form an organo-lead complex, which then will probably sorb to soil particles. Lead sorption

increases over the pH range of approximately 5-8, and above pH 6, it becomes the dominant process.

MANGANESE

Manganese (Mn) can occur in all valence states from -3 to +7. Mn(II) is very common and forms many salts. The solubility of manganese depends upon the pH of the aquatic environment. Mn(III) and Mn(IV) are only slightly soluble, though under neutral pH conditions these species may be reduced to Mn(II) which is more soluble and more mobile. Manganese can be chelated by a variety of organic and inorganic ligands. These ligands tend to keep manganese in solution. The soluble fraction of manganese ranges from 15-95 percent of the total, and is not dependent on pH, alkalinity, specific conductivity, or concentration of humic substances in water (Laxen et al., 1984). Soil-bound manganese can be dissolved by organic acids (Pohlman and McCall, 1986).

MERCURY

Mercury (Hg) can be found in three oxidation states in the environment. Elemental mercury, Hg(0), is a liquid at ordinary temperatures. Mercurous mercury, Hg(I), occurs primarily as Hg_2^{2+} under environmental conditions and does not form hydroxides, oxides, or sulfides. Mercuric mercury, Hg(II), forms stable complexes with common ligands. Some of these complexes are fairly soluble while others are quite insoluble (EPA, 1979). Mercuric hydroxide is not found in aquatic systems, but mercuric oxide is found and is soluble. In the presence of sulfide ion, mercuric sulfide (HgS) will precipitate from solution (Cotton and Wilkinson, 1982).

Elemental mercury is the predominant species in a moderately oxidizing environment above pH 5. Mercury becomes more soluble in the presence of chloride. Under mildly reducing conditions, HgS, which has an extremely low water solubility, will form (EPA, 1979).

The dominant process controlling mercury transport in the aquatic environment is sorption to particulates and sediments. The binding capacity of the sediment is related to its organic content; pH does not affect the sorption process. Desorption does not occur readily; therefore, mercury will tend to accumulate in sediments (Ramamoorthy and Rust, 1978).

Mercury can undergo microbial alkylation to methyl or dimethyl mercury in anaerobic or aerobic sediments. Organic forms of mercury exist in dynamic equilibrium with inorganic forms of mercury in natural waters. Methylation is of extreme importance in the environmental fate of mercury, because methylated mercury compounds are more water soluble, more easily absorbed through biological membranes, and bioaccumulated within animal tissues to a far greater extent than inorganic mercury. No measurements for alkyl mercury have been made at the NAS Alameda site; if mercury is detected in the lagoon sediments, it is likely that some fraction will be in the organic form.

Mercury has a high vapor pressure, and volatilization is a significant transport pathway for the metal. The rate of volatilization of mercury and its inorganic compounds from aquatic systems decreases in the order Hg> Hg_2Cl_2 > $HgCl_2$ > HgS > HgO. In soils, volatilization was found to increase with temperature and alkalinity (EPA, 1979).

Mercury's mobility in soils depends on the soil environment. In a study of the binding of trace metals to eleven soils, mercury was found to be moderately to highly mobile (Korte et al., 1976). The clay content of soils does not significantly affect sorption (Kabata-Pendias and Pendias, 1984). Baes et al. (1984) has reported a K_d of 10 mL/g for inorganic mercury.

NICKEL

Nickel (Ni) is found in the +2 oxidation state in the environment. Nickel forms complexes with hydroxide, nitrate, sulfate, and humic and fulvic acids. These complexes are moderately soluble and, at pH below 9, can result in Ni^{+2} concentrations greater than 60 μg/liter. Nickel hydroxide or

carbonate will precipitate from solution above pH 9 (Cotton and Wilkinson, 1982; EPA, 1979).

Humic materials complex with nickel to form soluble complexes. Solutions of humic acid and nickel carbonate solubilize nickel. In the presence of nickel sulfide, humic acid did not release nickel (EPA, 1979). The nickel-humic complex is stable enough to inhibit the precipitation of nickel hydroxide, carbonate, or sulfide (EPA, 1979).

There are few controls on nickel's mobility in the environment. Nickel can complex with commonly occurring inorganic ligands; however, these complexes are fairly soluble. Organic matter can complex with nickel, increasing the metal's mobility. Solid organic matter can sorb nickel, as can metal oxides (Kabata-Pendias and Pendias, 1984) and clay (Korte et al., 1976). The K_d of 150 mL/g reported by Baes et al. (1984) will be used in this assessment.

SELENIUM

The oxidation state of selenium (Se) will influence its mobility. Selenium can be found in the -2, 0, +4, and +6 oxidation states. Under oxidizing conditions over a wide pH range, selenium will be found in the +4 or +6 oxidation state as oxyanions and organo-selenium compounds. In aquatic environments $HSeO_3^{-1}$, SeO_3^{-3}, and SeO_4^{-2} are found (EPA, 1979). Elemental selenium can also be formed over a wide pH range under mildly oxidizing to reducing conditions (Pourbaix, 1963).

In soils, pH and E_h will control selenium mobility. In acidic soils and soils with high organic matter content, the largely immobile selenides and selenium sulfides are found. In neutral, well-drained, mineral soils, selenites (Se(IV)) are found; some metal selenites, which are soluble, may also be found. In addition, selenites, as $HSeO_3^{-1}$ or SeO_3^{-2}, can be sorbed by iron oxides and removed from solution. In alkaline, well-oxidized soils,

selenates (Se(VI)) will dominate. Selenates are very soluble, not well sorbed to soil particles, and hence very mobile (Kabatas-Pendias and Pendias, 1984).

Selenium sorption will be dependent upon the characteristics of the soil in which it is found. As could be expected from the above description, pH is an important variable. Frost and Griffin (1977) studied the effect of pH on selenium in landfill leachate with respect to sorption on clay materials; at high pH, they found Se(IV) was very mobile. Mobility decreased as pH decreased to pH 2 and then increased again; $HSeO_3^{-1}$ was the predominant species sorbed. Fuller (1978) reported similar results for metals sorbed as anion, which were found to be more easily mobilized than compounds sorbed as cations.

Korte et al., (1976) found that the sorbing action of clays removed selenium very effectively from solution. They found selenium mobility was correlated to clay content, surface area, free ion oxide content, and pH.

SILVER

Silver (Ag) can be found in the +1, +2, or +3 oxidation states; the dominant oxidation state is Ag(I). The environmental chemistry of silver is governed primarily by the solubility of its salts. Under the Eh-pH conditions commonly found in aquatic systems, metallic silver can be formed (Pourbaix, 1963). Silver metal has a very low solubility and this reaction can therefore control the level of silver in solution (EPA, 1979). Various silver salts can also control silver's solution concentration.

Cations and anions of silver occur in soils, but generally do not affect the immobility of silver unless the pH falls below 4 (Kabata-Pendias and Pendias, 1984). Sorption to manganese oxide is a major factor controlling silver mobility. Ferric oxides and clays can also sorb silver, but to a lesser extent (Kharka et al., 1968). In the presence of soil organic matter, silver mobility is fairly low. Silver has been observed to be mobilized by

cation exchange reactions, but only when H^+ is the competing cation, for example, at low pH (Kahn et al., 1982).

Silver mobility in groundwater was determined using a thermodynamic equilibrium model (Brown et al., 1986). The results of the modeling showed that nearly all the silver present in groundwater was precipitated. Under reducing conditions in the presence of sulfide, Ag_2S would precipitate. As the Eh was raised to oxidizing conditions, formation of bisulfide (HS^{-1}) kept silver concentrations very low.

Gerritse et al. (1982) reported a K_d of 10,000 ml/g for silver in both anaerobically digested sewage sludge and aerated sewage sludge. When the sludge solutions were placed in sandy loam or sandy soils, the K_ds for silver were reported as 250 mL/g and 100 mL/g, respectively. When inorganic solutions were equilibrated with sandy loam or sandy soils, the K_ds were approximately 1,000 mL/g and 750 mL/g, respectively. Baes et al. (1984) reported a K_d of 45 mL/g. Based on the environmental chemistry of silver, it is likely that the distribution coefficient is large (i.e., most silver is associated with soil or is in a precipitated form in water).

THALLIUM

The behavior of thallium (Tl) in the environment has not been widely studied. Thallium has been found to sorb onto clays and, under reducing conditions, precipitate as the sulfide. Under aerobic conditions, thallium can form soluble salts, which increases its mobility in water (EPA, 1979).

Thallium can be found in either the +1 or +3 oxidation states. Tl(I) is more stable in water than is Tl(III). Tl(III) can form organometallic complexes, while Tl(I) forms complexes only with oxygen, sulfur, and halogen ligands (Cotton and Wilkinson, 1982). Tl(I) is the dominant oxidation state under mildly oxidizing to reduced conditions. Under very reducing conditions, thallium may be precipitated as the metal, or in the presence of sulfur, as the sulfide (EPA, 1979).

Montmorillonite clays have been found to sorb thallium (Magorian et al., 1974). Adsorption was more effective at pH 8.1 than at pH 4. Under basic conditions, thallium was found to sorb onto ferric oxide. Either of these types of interactions is likely to occur in soils. Because thallium is not a well-studied element, it is difficult to determine the behavior of thallium in the environment. Thallium is likely to be mobile in solution, but adsorption to soil particles may also occur.

Thallium's metallic and covalent radii are similar to those of lead. In addition, the univalent ionic radius of thallium is similar to those of potassium and rubidium. Therefore, it is likely to thallium will behave like lead, potassium, or rubidium depending on pH, Eh, and the presence of other ligands.

TIN

Tin (Sn) occurs in both the +2 and +4 oxidation states in natural materials. Sn(II) is present only in highly acid and reducing environments (Kabata-Pendias and Pendias, 1984). The element is known to form anionic hydroxides and oxides, and to form complexes with organic materials. Tin may also form covalent bonds with organic chemicals; the manmade organotin chemical tributyltin is an example. The mobility of tin in soils is greatest at low pH. Tin is readily taken up by plants, and may be concentrated in roots and other plant parts (Kabata-Pendias and Pendias, 1984). Tin may be accumulated in mammalian tissues (Versar, 1975).

VANADIUM

The environmental chemistry of vanadium is very complex. Vanadium (V) can be found in solution in four different oxidation states: V(II), V(III), V(IV), and V(V). V(II) is not commonly found in aquatic or soil systems. Vanadium is relatively mobile in neutral and alkaline soils. V(V) is more soluble and more mobile than V(IV) or V(III) (Vinogradov, 1959). A major

factor in controlling vanadium's mobility is pH. Vanadium tends to precipitate at higher pH levels.

ZINC

Zinc (Zn) is found in the +2 oxidation state in the environment. The soil chemistry of zinc is controlled by the pH of the soil. In acidic soils, zinc adsorption is related to the density of cation exchange sites; in alkaline soils, the chemistry is dominated by organic ligands. Competition for binding sites by other metal cations may lead to mobilization of adsorbed zinc. In alkaline soils, zinc can form organo-zinc complexes, which would increase the metal's mobility (Kabata-Pendias and Pendias, 1984). Metal oxides and clays also influence zinc's mobility in soils. Zinc was found to be highly associated with oxides and could be adsorbed by clay minerals. Soils that contain high levels of calcium and phosphorus will also immobilize the metal (Kabata-Pendias and Pendias, 1984).

Zinc can be found in water as a hydrated ion, a metal-organic complex, or a metal-inorganic complex. Zinc forms complexes with humic and fulvic acids, which can increase its solubility (Ong et al., 1970; Rashid and Leonard, 1973). It was found that these complexes may keep zinc in solution at a pH as low as 3. Zinc can also precipitate as the sulfide, hydroxide, or oxide, depending on the redox conditions in the water (Pourbaix, 1963). One study showed that iron oxides and phosphates removed zinc from river water (Honba et al., 1983).

RADIONUCLIDES

A large number of radioactive elements can potentially be present at waste sites. Sources of radioactivity which are frequently encountered at waste sites include naturally occurring isotopes (e.g., radon), medical wastes (e.g., radioisotopes of hydrogen, iodine, and phosphorus), and products of ore refining (e.g., uranium). The various radionuclides vary greatly with respect to their chemical/physical properties, radioactive half-lives, and proportions

of alpha, beta, and gamma radiation emitted. The nature of the radioactive materials at the NAS Alameda site has not been determined; therefore, no conclusions can be made as to the potential for these materials to migrate from their point of release.

4.2 MECHANISMS OF MIGRATION

There are a number of mechanisms by which chemicals of potential concern may migrate from contaminated areas to on-site or off-site receptors. Migration of chemicals to groundwater may occur by percolation of infiltrating rainwater or by bulk flow of chemicals released from underground storage containers. Transport to the Seaplane Lagoon and Oakland Estuary may occur via surface water runoff from contaminated soil, via airborne deposition of contaminants, via stormwater collected in the drainage system at the site, or via groundwater discharge. Migration of chemicals to air may occur via fugitive dust emissions or volatilization. Chemical vapors and landfill gases could also migrate through permeable zones or utility lines. The potential for the chemicals of potential concern to migrate via these mechanisms is described in the following subsections.

4.2.1 Migration into Air

Fugitive dust emissions of contaminated particulates could occur in areas of the site that are not completely paved or covered with vegetation, such as the Cans C-2 area, Building 389, West Beach Landfill, and the 1943-1956 Disposal Area. Particulate emissions would be of potential concern primarily for inorganic chemicals and for those organic chemicals which are relatively nonvolatile, such as PCBSs insecticides, herbicides, and phthalates. Emissions of asbestos-containing wastes would be of potential concern if the layers of soil currently covering contaminated wastes were to be disturbed, such as during remediation.

Volatilization of chemicals from surface soil and from shallow groundwater is a pathway of potential concern for areas of the NAS Alameda

site where organic solvents or petroleum products are present. Areas where volatilization may be of concern include: Buildings 360, 400, 410, 530, the Cans C-2 area, the West Beach Landfill, and the 1943-1956 Disposal Area. The shallowness of the water table and sandy consistency of the fill used to construct the site create a likely route of migration from any groundwater plume to the ambient air. Chemicals released by volatilization might undergo subsequent degradation and would be diluted and transported in the air; the direction of movement and degree of dilution before reaching a receptor have not been determined.

There is evidence that explosive gases and fuel vapors may be present in several of the areas on-site, including Buildings 459 and 547, Area 97, the Power Plant and former Oil Refinery, the NAS Alameda sewer system, the West Beach Landfill, and the 1943-1956 Disposal Area. Due to the heterogeneous nature of the fill making up the NAS Alameda site, the direction of flow of chemical vapors and landfill gases would likely be determined by local features of the subsurface, such as permeable zones (buried utility lines, sewers) or areas of impermeable soil cover (paving, buildings). These gases may ultimately discharge to the soil surface and become mixed with the ambient air, or may seep into buildings.

4.2.2 Percolation into Groundwater

The groundwater transport potential of each of the chemical classes of potential concern at the NAS Alameda site was discussed in Section 4.1. Areas where chemicals are known or suspected to be present in subsurface soils or groundwater include Area 97, the Cans C-2 area, Buildings 360, 301 and 389, the West Beach Landfill, and the 1943-1956 Disposal Area. The extent to which these chemicals will actually migrate within the shallow aquifer depends on a number of site-specific factors which have not been fully determined at the NAS Alameda site. The factors include the direction and rate of groundwater flow, the physical and chemical makeup of the saturated and unsaturated soils beneath the site, and the geochemistry and microbiology of the subsurface environment. Many of the organic chemicals of potential concern are

susceptible to rapid chemical or biological transformation in soils, and therefore may not reach groundwater. The solubilities of the inorganic chemicals of potential concern may vary depending on the pH and redox conditions prevailing in the area.

4.2.3 Migration in Surface Water

There are several possible routes of migration to surface water at the NAS Alameda site. One such route involves dissolution or suspension of contaminants in surface runoff flowing into the storm drains at the site. The drain system was used to discharge industrial wastewater until 1975, but now discharges only storm water. The integrity of the storm drain system is unknown, and it is possible that some contaminants may enter the system from surrounding soils, since the drain system is mostly set below the water table, and any flow might be into the drains rather than out into the soil. Areas which may potentially contribute to surface water contamination via the storm drain system include the West Beach Landfill, the 1943-1956 Disposal Area, Area 97, and the Fire Training Area.

Another possible route of migration to the marine environment is via discharge of groundwater from the site into the Seaplane Lagoon or Oakland Estuary. The hydrogeology of the site has not been studied with sufficient detail to determine the location or volume of discharge.

If discharged into the lagoon or estuary, contaminants would partition between the water, sediment, air, and biota compartments of the aquatic system. Hydrophobic organic chemicals such as PCBs and phthalates would tend to concentrate primarily in sediments, although the presence of dissolved or suspended organic material in the water can increase the solubility of these chemicals considerably. The transport and fate of chemicals in a marine environment is difficult to estimate, as it may depend on a large number of physical, hydrological, and chemical factors. Processes which would effect the concentrations to which aquatic organisms might be exposed include:

mixing, sedimentation, bioturbation, adsorption/desorption, precipitation/dissolution, and volatilization.

Chemicals of potential concern may potentially also migrate to the wetlands near the Seaplane Lagoon and West Beach Landfill. Routes of migration which could lead to contamination of these wetlands include deposition of airborne particulates, dissolution of vapors seeping through the soil, and inflow of chemicals in surface runoff.

4.2.4 Uptake of Inorganic Contaminants by Plants

Vegetables grown in the vicinity of the NAS Alameda site could potentially become contaminated as a result of irrigation with contaminated water, growth in contaminated soils, or deposition of airborne particulates on foliage. Metals may be translocated into plant tissues either by active transport or by passive partitioning. Baes et al. (1984) estimated uptake factors for many elements, and reported that uptake by the vegetative parts (leaves) of the plant exceeded uptake by the reproductive and storage parts of plants (fruit, seeds, roots). Metals of particular concern for plant uptake are cadmium and lead. Certain organic chemicals may be taken up by active transport mechanisms within the plant; however, most organics are accumulated via passive diffusion and partitioning into plant tissues.

Direct deposition of chemicals onto vegetables would be of particular concern for leafy vegetables, such as lettuce or broccoli, since they would be expected to intercept a relatively high percentage of settling particulates. Chemicals which would be of concern for deposition onto vegetables are metals and semi-volatile organics such as PAHs, PCBs, and pesticides.

4.3 DEMOGRAPHICS

Land use in the vicinity of NAS Alameda is primarily residential and military. Approximately 5,300 civilian and 2,700 military personnel work or live at NAS Alameda. A total of 1,213 residential units are located on the

base, housing children and adults. These residential units are in the northeast portion of the base, away from the industrial activities on NAS Alameda. Common on-base recreational activities for NAS Alameda residents include picnicking, running, and target practice (DHS, 1988a).

To the north of NAS Alameda, across the Oakland Inner Harbor Channel, is the Naval Supply Center-Oakland (NSCO). NSCO is one of the largest naval supply centers on the West Coast, occupying 541 acres of land. The land east of the station is used for industrial, residential, and public purposes. The 81-acre Naval Supply Center Oakland-Alameda Annex is located immediately east of NAS Alameda. The Todd Shipyards are located adjacent to the northeast corner of NAS Alameda on the Oakland Inner Harbor Channel. The shipyards consist of dry dock facilities which are used for repairing ships (E & E/WESTDIV, 1983). To the east of Todd Shipyards is the 107-acre Naval Supply Center Oakland-Alameda Facility (NSCO-AF). Bordering the eastern side of NSCO-AF is the College of Alameda Junior College which has a current enrollment of about 5,100 students.

The remaining land use east of NAS Alameda consists of residential parcels with scattered commercial establishments such as restaurants and retail stores. There are a number of schools located in this residential area including Woodstock Elementary School with 602 students, Chipman Elementary School with 575 students, Longfellow Elementary School with 487 students, George P. Miller Elementary School with 462 students, William G. Paden Adult School, and Encinal High School, located adjacent to the southeast corner of the station, with 1,050 students. Located east of Encinal High School is the Robert Crown Memorial State Beach where swimming and windsurfing are popular activities. Crab Cove, a State-protected marine reserve, is located on the western end of this beach (E & E/WESTDIV, 1983; DHS, 1988a). Other sensitive targets such as day care facilities and convalescent homes, will be identified in the final PHEE report.

4.4 POTENTIAL HUMAN EXPOSURE PATHWAYS

As discussed previously, many individuals live or work on NAS Alameda and the surrounding area. These individuals may be potentially exposed to contaminants through a number of exposure pathways. In order for a chemical to pose a human health risk, a complete exposure pathway must be identified. A complete exposure pathway consists of four elements: 1) a source and mechanism of chemical release to the environment, 2) an environmental transport medium (e.g., air, soil) for the released chemical, 3) a point of potential human contact with the contaminated medium (known as the exposure point), and 4) a human exposure route (e.g., inhalation) at the contact point (EPA, 1986f). This current PHEE analysis will be developed from two different perspectives. The first will consider current land use in the area and the second will consider potential future uses that may differ from the current situation.

In this section, exposure pathways are discussed for each of the study areas of potential concern at NAS Alameda. Potential exposure pathways are evaluated for each potentially contaminated environmental medium: soil, groundwater, surface water, and air.

4.4.1 Current-Use Conditions

As discussed in Section 4.3, NAS Alameda is an operating naval facility employing both military and civilian personnel. Military personnel and their families live on NAS Alameda. Individuals with access to the base may also engage in recreational activities such as jogging or picnicking on the facility. Due to the security at the facility, trespassers are not expected. Discussed below are potential current-use exposure pathways for contaminants in soil, groundwater, surface water, and air. Table 4-3 evaluates the current-use exposure pathways.

Table 4-3

Human Exposure Pathways for NAS Alameda
Under Current-Use Conditions

Exposure Medium	Exposure Point	Potential Receptors	Route of Exposure	Pathway Complete?
Soil	Fire Training Area	Workers	Direct Contact	Yes. Workers may come in contact with contaminated soil while fire fighting.
	Building 360	Workers	Direct Contact	Yes. Workers in crawl spaces may be exposed.
	Building 389	Workers	Direct Contact	Yes. Surface soils have had PCBs detected.
	Cans C-2 Area	Workers	Direct Contact	Yes. Workers have access to contaminated soils.
	1943-1956 Disposal Area	Workers	Direct Contact	No. Workers in this area work in paved areas.
		Residents/ Picnickers	Direct Contact	Yes. All individuals with access to the base may picnic here and come in contact with contaminated soils.
	West Beach Landfill	Workers	Direct Contact	No. The site is kept locked by NAS Alameda security with limited access and workers generally stay on the road along the dike surrounding the landfill.
Groundwater				No. There are no identified users of groundwater.

Table 4-3
(continued)

Human Exposure Pathways for NAS Alameda
Under Current-Use Conditions

Exposure Medium	Exposure Point	Potential Receptors	Route of Exposure	Pathway Complete?
Surface Water	Seaplane Lagoon	Individuals Fishing	Ingestion of Fish	Yes. Contaminants detected in sediments may be absorbed by fish.
	Oakland Estuary	Boaters	Direct Contact with Sediments	No. Boaters are unlikely to contact sediments.
		Swimmers	Direct Contact with Sediments	No. Swimmers are not expected in this area.
Air	Buildings 459 and 547	Workers	Subsurface Vapor Migration	Yes. Subsurface vapors have been detected in underground lines.
		Residents	Subsurface Vapor Migration	No. Vapors have not been detected in basements or crawl spaces.
	Area 97	Workers	Subsurface Vapor Migration	Yes. Subsurface vapors have been detected in underground lines.
		Residents	Subsurface Vapor Migration	No. Vapors have not been detected in basements or crawl spaces in houses.

Table 4-3
(continued)

Human Exposure Pathways for NAS Alameda
Under Current-Use Conditions

Exposure Medium	Exposure Point	Potential Receptors	Route of Exposure	Pathway Complete?
Air (continued)	Oil Refinery	Workers	Subsurface Vapor Migration	Yes. Vapors have been released from this area in the past and may be present in underground lines.
	Fire Training Area	Workers	Airborne Particulates	Yes. Workers in this area may be exposed during fire training activities.
	Fire Training Area	Recreational Users of Oakland Estuary	Airborne Particulates	Yes. Boaters may be exposed to airborne particulates.
	Building 360	Workers	Passive Diffusion	Yes. Volatile soil contaminants may be released into the work area.
	Buildings 301 & 389	Workers	Airborne Particulate	Yes. Site is unpaved and particulates may be released during site activities.
		Residential Users of Oakland Estuary	Airborne Particulate	Yes. Boaters may be exposed to airborne particulates.

Table 4-3
(continued)

Human Exposure Pathways for NAS Alameda
Under Current-Use Conditions

Exposure Medium	Exposure Point	Potential Receptors	Route of Exposure	Pathway Complete?
Air (continued)	CANs C-2 Area	Workers	Airborne Particulate Passive Diffusion	Yes. Site activities may result in particulate generation and release of volatile contaminants.
		Residents east of NAS Alameda	Airborne Particulate Passive Diffusion	Yes. Residents adjacent to the CANs C-2 Area may be exposed to particulates and volatiles.
	Sewer System	Workers & Residents	Vapor Migration	No. No information is available to predict direction of migration.
	1943-1956 Disposal Area	Workers	Airborne Particulate	Yes. Activities may lead to dust generation.
			Passive Diffusion	Yes. Site is unpaved.
			Vapor Migration	No. Information is not available on possible methane production.
	West Beach Landfill	Workers	Airborne Particulate	No. Dust generating activities do not occur.
			Passive Diffusion	Yes. Site is unpaved.

Table 4-3
(continued)

Human Exposure Pathways for NAS Alameda
Under Current-Use Conditions

Exposure Medium	Exposure Point	Potential Receptors	Route of Exposure	Pathway Complete?
Air (continued)			Vapor Migration	No. Information is not available on methane production.

4.4.1.1 Soil Contaminants

As discussed in Section 2.3, heavy metals and other contaminants have been detected in surface soils from the Building 360, Building 389 and the Cans C-2 Area. Other areas have not been investigated for possible surficial soil contamination. Potential routes of exposure to contaminated soils include direct soil contact and inhalation of contaminated airborne particulates. The direct contact pathway will be discussed in this section and in Section 4.4.2.1. Inhalation of airborne particulates will be discussed under the air pathways in Section 4.4.1.4.

Under current-use conditions, individuals working or living at NAS Alameda may be exposed to soil-borne contaminants via direct contact with contaminated soils. Direct contact with contaminated soil can lead to exposure either via inadvertent or intentional soil ingestion or via dermal absorption. The direct ingestion of contaminated soil is especially of concern for young children, who may ingest contaminated dirt by normal mouthing of soiled objects or their fingers and/or hands (Mahaffey, 1978; LaGoy, 1987; CDHS, 1987). Older children are less likely to eat soil or to mouth soiled objects, but they still may ingest dirt from their hands. Adults on site may ingest some contaminated soil, but they are less likely to be exposed by this route, unless they have a high incidence of hand-to-mouth contact (e.g., smokers, gardeners).

Individuals may come in contact with contaminated surface soils while jogging, or in connection with excavation activities. A former picnic area is located between two landfills. At this time, the picnic area is not in use, although access is not restricted and picnic tables are still in place (Cates, 1988). If this area were to be used, picnickers could be exposed intermittently while picnicking or playing. Workers could contact deeper soil layers as well as surface soils during occupational activities.

At this time, the direct contact exposure pathway is identified as relevant for the following areas with unpaved potentially contaminated soils:

Cans C-2 Area, Fire Training Area, Building 389, and the 1943-1956 Disposal Area. When further analytical data are available, additional study areas may be considered for evaluation of this route.

4.4.1.2 Groundwater Contaminants

As mentioned in Section 2.3, pesticides, metals, various organic solvents, PAHs, and petroleum hydrocarbons have been detected in shallow groundwater underlying NAS Alameda. At this time, there are no identified users of groundwater underlying NAS Alameda. Groundwater east of the base is used for irrigation by the City of Alameda and private well owners. Potential hydrological interconnection of these aquifers to the groundwater underlying the site will be addressed in the RI. At this time, no information regarding interconnection is available. Therefore, under current-use of groundwater, the exposure pathway is incomplete.

In the future, wells may be located in areas affected by multiple sources of contamination from the different study areas. Any of the areas with known or suspected soil contamination could potentially be sources for groundwater contamination.

4.4.1.3 Surface Water Contaminants

Activities in the Seaplane Lagoon and Oakland Estuary are the primary exposure routes for potential surface water contaminants. Individuals may also be exposed to water ponding on contaminated soil or to surface water runoff from areas with surface soil contamination. At this time, no data are available to evaluate these latter two secondary pathways. For purposes of this preliminary PHEE, only exposures to contaminants in the Seaplane Lagoon and Oakland Estuary will be discussed.

As discussed in Section 2.1, waste water was discharged directly to the Oakland Estuary and the Seaplane Lagoon in the past. At this time, no surface water samples have been analyzed from the Oakland Estuary or Seaplane Lagoon;

however, sediment data from these areas indicate the presence of heavy metals in both areas. Humans may be exposed directly to surface water contaminants through recreational activities such as boating and wind-surfing, and indirectly via the consumption of contaminated seafood. Humans generally are not frequently exposed to sediments. However, consumption of fish that eat organisms living in contaminated sediments could potentially be an indirect human exposure pathway.

Currently, the Seaplane Lagoon is used for helicopter exercises and for recreational fishing. All individuals who have access to the base have access to the Seaplane Lagoon fishing pier. It is unknown how many people fish from this pier.

Individuals may be exposed indirectly through the consumption of fish in contact with surface water and/or sediments. Fish caught in the Seaplane Lagoon have been reported as smelling "oily" (E & E/WESTDIV, 1983). However, no biota or water samples have been collected from the lagoon and therefore, potential contaminant exposures through this route cannot be assessed.

As mentioned in Section 4.2, recreational boating and commercial shipping occur in the Oakland Estuary and Inner Harbor. East from NAS Alameda is a large residential population which lives in houseboats in the harbors. These individuals could potentially be exposed to contaminated surface water. At this time, no data are available which indicate the presence of surface water contaminants.

4.4.1.4 Air Contaminants

No data currently exist to quantify the concentrations of potential airborne contaminants. However, as discussed in Section 4.2, the air route can be an important mechanism of release to the environment for surface and subsurface soil contaminants. Contaminants which may be transported via air include chemicals adsorbed to surface soil particles, volatile organics that passively diffuse through subsurface soils, and volatiles released to air

through bulk flow of gases generated through biodegradation of subsurface contaminants or from underground leaks (e.g., methane, petroleum hydrocarbons).

Most locations where surface soil contaminants have been detected are either paved or vegetated. However, the Cans C-2 Area, Building 389, the Fire Training Area and the landfills contain unpaved areas which may provide routes for airborne contaminant release.

As discussed in Section 2.3, volatile organic and inorganic contaminants as well as potential particulate contaminants have been found in surface and subsurface soils. Both particulates and volatiles could be released from these areas resulting in exposure to workers and residents.

Another current-use exposure pathway may result from landfill gas or gas generated from petroleum hydrocarbons traveling through subsurface soils. Bulk transported gases often accumulate in confined spaces, such as basements. Individuals living in houses with basements or crawl spaces may be exposed via this route. Workers working near underground lines or in underground rooms may also be exposed to contaminants via this route.

4.4.2 Future-Use Conditions

At this time, the Navy has no plans to change land-use at NAS Alameda in the future. However, in the remote future if NAS Alameda were to close, land-use activities at NAS Alameda may change. Hypothetical potential future-uses of the facility may include increased residential areas and parks. Pavement and vegetation may be removed, providing increased potential for environmental release for surface and subsurface contaminants. In this section, potential future uses of NAS Alameda are discussed and the resulting exposure pathways evaluated for soil, groundwater, surface water and air. Table 4-4 evaluates potential future exposure pathways.

Table 4-4

Human Exposure Pathways for NAS Alameda
Under Future-Use Conditions

Exposure Medium	Exposure Point	Potential Receptors[a]	Route of Exposure	Pathway Complete?
Soil	Building 41	Workers	Direct Contact	Yes. If pavement removed.
	Building 10	Workers	Direct Contact	Yes. If pavement removed.
	Area 97	Workers	Direct Contact	Yes. If pavement removed.
	Fire Training Area	Workers	Direct Contact	Yes. If pavement removed.
	Building 114	Workers	Direct Contact	Yes. If pavement removed.
	Building 5	Workers	Direct Contact	Yes. If pavement removed.
	Building 360	Workers	Direct Contact	Yes. If pavement removed.
	Building 410	Workers	Direct Contact	Yes. If pavement removed.
	Building 400	Workers	Direct Contact	Yes. If pavement removed.
	Building 530	Workers	Direct Contact	Yes. If pavement removed.
	Building 301	Workers	Direct Contact	Yes. If pavement removed.

a Residents are unlikely receptors unless NAS Alameda is closed.

Table 4-4
(continued)

Human Exposure Pathways for NAS Alameda
Under Future-Use Conditions

Exposure Medium	Exposure Point	Potential Receptors[a]	Route of Exposure	Pathway Complete?
Soil (cont'd)	Building 389	Workers	Direct Contact	Yes. If pavement removed.
	Cans C-2 Area	Workers	Direct Contact	Yes. If pavement removed.
	Yard D-13	Workers	Direct Contact	Yes. If pavement is removed.
	Building 459	Workers	Direct Contact with Subsurface Soils	Yes. During excavation or remediation activities.
	Building 547	Workers	Direct Contact with Subsurface Soils	Yes. During excavation or remediation activities.
	Oil Refinery	Workers	Direct Contact with Subsurface Soils	Yes. During excavation or remediation activities.
	CANs C-2 Area	Workers	Direct Contact with Subsurface Soils	Yes. During excavation or remediation activities.

a Residents are unlikely receptors unless NAS Alameda is closed.

Table 4-4
(continued)

Human Exposure Pathways for NAS Alameda
Under Future-Use Conditions

Exposure Medium	Exposure Point	Potential Receptors[a]	Route of Exposure	Pathway Complete?
Groundwater				No. At this time no information is available regarding ground water uses to evaluate this pathway.
Surface Water	Seaplane Lagoon	Individuals Fishing	Ingestion of Fish	Yes. Same as Current-Use.
	Oakland Estuary	Boaters	Direct Contact with Sediments	No. Same as Current-Use.
		Swimmers	Direct Contact with Sediments	No. Same as Current-Use.
Air	Building 41	Workers	Airborne Particulate Passive Diffusion	Yes. If pavement is removed.
	Building 459	Workers	Passive Diffusion	Yes. If pavement is removed.
	Building 547	Workers	Bulk Transport	Yes. If pavement is removed.

a Residents are unlikely receptors unless NAS Alameda is closed.

Table 4-4
(continued)

Human Exposure Pathways for NAS Alameda
Under Future-Use Conditions

Exposure Medium	Exposure Point	Potential Receptors[a]	Route of Exposure	Pathway Complete?
Air (cont'd)	Building 10	Workers	Airborne Particulates	Yes. If pavement is removed.
	Area 97	Workers	Airborne Particulate	Yes. If pavement is removed.
		Residents	Bulk Transport	Yes. Same as current-use.
			Passive Diffusion	Yes. If pavement is removed.
	Oil Refinery	Workers	Airborne Particulate	Yes. If pavement is removed.
		Residents	Bulk Transport	Yes. Same as current-use.
	Oil Refinery	Residents	Passive Diffusion	Yes. If pavement is removed.
	Fire Training Area	Workers	Airborne Particulate	Yes. Same as current-use.
		Boaters	Airborne Particulate	No. If pavement is removed.

a Residents are unlikely receptors unless NAS Alameda is closed.

Table 4-4
(continued)

Human Exposure Pathways for NAS Alameda
Under Future-Use Conditions

Exposure Medium	Exposure Point	Potential Receptors[a]	Route of Exposure	Pathway Complete?
Air (cont'd)	Building 114	Workers	Airborne Particulate Passive Diffusion	Yes. If pavement is removed.
	Sewer System	Workers	Passive Diffusion Bulk Transport	No. Information on gas transport is incomplete.
	1943-1956 Disposal Area	Residents	Bulk Transport	No. Information on methane generation is incomplete.
	Yard D-13	Workers	Airborne Particulate Passive Diffusion	Yes. If pavement is removed.

a Residents are unlikely receptors unless NAS Alameda is closed.

4.4.2.1 Soil Contaminants

In the absence of any remediation or restrictions, under future land-use conditions, access to known contaminated areas may increase (e.g., increased use of picnic areas) and therefore, more individuals may potentially be exposed to soil contaminants. Workers may have additional short-term exposure to soil contaminants during excavation activities.

4.4.2.2 Groundwater Contaminants

It is suspected that some, if not all, of the contaminated groundwater beneath NAS Alameda is located in perched saline layers that are not connected with potable groundwater aquifers. Insufficient hydrogeologic information is available to evaluate the potential for future domestic use of area groundwater. Groundwater characteristics will be investigated in the RI to determine if any potential groundwater receptors exist.

4.4.2.3 Surface Water Contaminants

The potential exposure pathways for surface water-borne contaminants are the same in the future as in the current-use scenario. In the future, greater access (i.e., if all portions of the population, both civilian and military, have access to the base) may be available to the Seaplane Lagoon fishing pier, resulting in a potentially larger exposed population. As the population of Oakland and Alameda increases, more people can also be expected to engage in recreational activities in the Oakland Estuary.

4.4.2.4 Air Contaminants

The contaminant release mechanisms discussed under current-use will be applicable under future-use conditions as well. Potentially exposed populations include residents, workers at NAS Alameda, and recreational users of Alameda. The removal of pavement or buildings may increase potential air

exposure. Conversely, the addition of pavement or buildings may decrease potential air exposures.

4.5 COMPLETE EXPOSURE PATHWAYS

In this section, each of the twenty identified study areas is evaluated with respect to the complete exposure pathways presented in Tables 4-3 and 4-4 and the data available to date. The likelihood of each pathway is discussed and site-specific receptors are identified.

4.5.1 Building 41

Currently, the area around Building 41 is paved; therefore, no exposure is anticipated at this time via direct soil contact or passive diffusion of volatile contaminants. No data are available regarding potential subsurface contaminants, therefore, potential migration to groundwater cannot be evaluated at this time.

In the future, however, pavement may be removed providing a route for release of potential surface and subsurface contaminants. The completed future pathways for Building 41 are: direct soil contact, inhalation of airborne particulates, and inhalation of organics volatilized from soils. Workers and potential on-site residents are the potential future receptors for the direct soil contact pathway and both airborne pathways. Off-site residents would be potential future receptors of airborne contaminants.

The future-use pathway for workers appears to be the most likely exposure scenario based on site history and future-use projections. The most probable air pathways will be identified based on RI sampling results.

4.5.2 Buildings 162, 459, and 547

As discussed in Section 2.1.1, these buildings are suspected of subsurface soil contamination due to potentially leaking underground tanks.

Petroleum hydrocarbon contamination is suspected in these areas. Therefore, under current and future use pathways these areas may contribute to subsurface vapor migration. However, Building 162 has no known history of underground tanks and therefore, it seems improbable that fuel-related contaminants will be present in the subsurface. Other activities at Building 162 may have contributed to subsurface contamination; however, this appears unlikely due to the presence of pavement and the small quantity of waste generated. Potential exposure via inhalation of volatile solvents or direct contact may occur; no data are currently available to evaluate this pathway. Until sampling data are available to indicate the presence of contaminants, all potential exposure pathways for Building 162 are incomplete.

As discussed in Section 2.1.1., underground tanks and fuel lines are reported to have leaked at both Building 459 and Building 547. Mobile constituents of these fuels from these tanks may potentially migrate via bulk transport of vapors or passive diffusion. Contaminants in waste oil (e.g., PAHs) may adsorb onto subsurface soil and present a direct soil contact exposure pathways for workers during excavation. Currently these sites are paved and therefore, exposure via the passive diffusion and direct contact pathways is unlikely.

Under current-use conditions, the only identified complete exposure pathway for Building 459 and 547 is bulk transport of hydrocarbon vapors. Potential receptors include workers at these service stations, residents living in areas north and east of Building 459, and residents living south and west of Building 547. Individuals predicted to have highest exposure to these contaminants are those workers with access to crawl spaces and residents with basements. At this time, no data are available to evaluate the possible extent of subsurface vapor migration.

Under potential future-use conditions, assuming that pavement has been removed, passive diffusion of volatile contaminants and bulk transport are identified as complete exposure pathways. The receptors for future-use are the same as identified under the current use scenario. In the future, workers

at the service stations would have the highest potential exposure to these contaminants. Since contamination in these areas is anticipated to be confined to subsurface soil, direct soil contact and airborne transport of particulates are incomplete pathways for Buildings 459 and 547.

4.5.3 Building 10 (Power Plant)

Subsurface soil contamination is suspected in the soils underlying Building 10, due to a history of underground storage tanks. Potential surface soil contamination is suspected due to a reported history of spills and leaks. The soils around Building 10 are currently paved. Subsurface migration of hydrocarbon vapors is not anticipated to be substantial due to the low volatility of diesel and bunker fuels and waste oil. Therefore, under current-use conditions, no complete exposure pathways have been identified.

Under future-use conditions, pavement may be removed resulting in potential direct soil contact and airborne particulate exposure to workers. Based on site history and future land-use projections, workers are the most probable future receptors of these contaminants. Off-site residents are not anticipated to be exposed frequently to contaminants from Building 10 due to the presence of buildings between this area and the site boundary.

4.5.4 Area 97

Fuel-related contaminants have been detected in surface and subsurface soils in Area 97. Currently, this area is covered with clean sod; therefore, direct contact with and airborne migration of soil contaminants are not likely. Although access to this area is not restricted to individuals on the base, use of this area as a potential recreation area is unlikely because it is located in the middle of a heavily-used traffic circle. Migration of subsurface contaminants has been demonstrated by reports of hydrocarbon vapors in nearby utility line ducts. Under current use, the vapor migration pathway is the only complete exposure pathway. Potential current receptors of vapor

borne contaminants include workers near Area 97 and off-site residents east of the base, as well as occasional visitors to the facility.

Under the future-use scenario, vegetation from this area may be removed, resulting in potential exposure to workers and residents via direct contact and airborne release of surface and subsurface contaminants. Airborne transport of contaminants is the most likely potential exposure pathway. Due to the prevailing wind pattern around NAS Alameda, workers and residents east of this area are the most probable receptors of airborne particulates and volatile organics released via passive diffusion from subsurface soils. Subsurface vapor migration patterns have not been established, but it is assumed that workers and residents are potential future receptors for this pathway. If access to this area is increased in the future, then exposure via direct soil contact is possible. Potential receptors via direct contact with soils include workers and residents. If current land usage practices continue, workers are the most probable future receptors for the direct contact pathway.

4.5.5 Oil Refinery

Subsurface contaminants have been detected in this area due the former operation of a disposal area for refinery waste. As discussed in Section 2.1.5, vapor releases at this area in the past have caused pavement to crack.

At this time, the site is paved. Under current-use conditions, only the vapor migration exposure pathway is complete. Workers in crawl spaces and off-site residents with basements may be exposed to vapor borne contaminants traveling from the oil refinery. At this time, workers are the more probable receptors.

In the future, pavement from this area may be removed resulting in a release mechanism for volatile contaminants. Volatile contaminants may be released via passive diffusion or through vapor transport. Potential future

receptors of these contaminants include on- and off-site workers and residents east of NAS Alameda. Workers excavating may be exposed via direct contact.

4.5.6 Fire Training Area

Surface soil contamination is suspected in the Fire Training Area. The site is unpaved and waste fuels are burned there regularly, resulting in airborne release of combustion products. Under current-use conditions, the direct soil contact and the airborne particulate exposure pathways are complete for workers. Workers are likely to be exposed via both routes; downwind residents and recreational users of the Oakland Estuary may potentially be exposed to downwind particulates.

Under future-use conditions, site activities may change, resulting in increased access to the site. On-base personnel may have access to this area and be exposed via direct contact. Children would be the most exposed receptors, due to their high incidence of soil ingestion. Airborne release of contaminants would be expected to continue under potential future-use conditions.

4.5.7 Building 114

Pesticides and other chemicals have been used and stored in Building 114. Currently, Building 114 is primarily an office building. The area is paved; no samples have been collected here.

At this time, no contaminant release to the environment is anticipated. However, in the future, pavement in this area may be removed resulting in potential exposure via direct soil contact and airborne contaminants: passively diffused volatile contaminants and airborne particulates. Workers may be exposed via direct soil contact. Receptors for airborne contaminants include on- and off-site workers, and off-site residents. Exposure is most likely for on-site workers involved in soil-disturbing activities such as landscaping or excavating.

4.5.8 Building 5

A variety of industrial activities are conducted at Building 5 resulting in the generation of metal plating wastes, paint stripping wastes and general maintenance wastes. No samples have been collected to determine if these wastes were released to soils in the area. Subsurface contamination is not anticipated because the building is located on a concrete floor. The site is currently paved. Therefore, under current-use conditions no complete exposure pathways have been identified.

In the future, pavement in this area may be removed resulting in potential release of contaminants in surface soils. Workers are the likely receptor population for direct contact. Workers and residents downwind from Building 114 are likely to be exposed to airborne contaminants released via dust generation and volatilization.

4.5.9 Building 360

Cyanides have been detected in surface soils underlying Building 360; metals and other electroplating related contaminants are suspected to also be present. Although the area surrounding Building 360 is paved, areas beneath the building are unpaved. Workers in crawl spaces beneath the building may be exposed to these contaminants via direct contact as well as by inhalation of volatile contaminants (e.g., cyanide). Exposures to workers in crawl spaces are anticipated to be intermittent and result in subchronic, or short term, exposure rather than long duration exposure. Therefore, under current-use, the direct contact and inhalation pathways are considered complete for intermittent exposure to workers.

Under future-use conditions, all pavement in the area may be removed and Building 360 removed, thereby providing for increased access to site contaminants. Workers in the area may potentially be exposed via direct soil contact and inhalation of volatile contaminants and airborne. Off-site

residents may also be exposed to airborne contamination from Building 360. If current land-use practices continue in the future, workers will be the most probable future receptors of site-related contaminants. Particulate transport is considered the more likely exposure pathway for Building 360-related contaminants because most of the contaminants are expected to be inorganic compounds which are likely to bind to soils rather than volatilize from soils.

4.5.10 Building 410

Paint stripping activities in Building 410 result in the generation of solvent and paint waste. No soil samples have been collected in this area; however, the potential exists for release of these wastes to the environment via spills and sewer leaks.

The site is currently paved; therefore, no release mechanism via surface soil or air has been identified. Under future-use conditions, pavement may be removed, providing a potential mechanism for contaminant release from surface and subsurface soils. Volatile chemicals may be released via passive diffusion; metals and paint particulates may be released during soil-disturbing activities. Workers may potentially be exposed to airborne contaminants.

4.5.11 Buildings 400 and 530

Missile rework activities have occurred in both Building 400 and Building 530; paint stripping wastes and metals are associated with this process. No sampling has occurred at either location and both locations are paved. Under current-use conditions, no environmental release mechanisms have been identified. Under future-use conditions, pavement at both of these areas may be removed. If so, workers may be exposed to surface soil contaminants via direct soil contact as well as via airborne release of contaminated dust and emission of volatile contaminants from the subsurface. Off-site residents east of the facility, and recreational users of the Seaplane Lagoon, may potentially be exposed to airborne contaminants. If current land-use patterns

continue in the future, workers are expected to be the only direct contact receptors.

4.5.12 Building 14

At this time, no environmental contamination source has been identified at Building 14. At one time, broken thermometers were reported to have spilled mercury on the second floor of the building; however, no contamination of environmental media is suspected as a result of this event. If the RI sampling identifies the presence of on-site contamination, then potential exposure pathways for Building 14 will be fully evaluated in the final PHEE.

4.5.13 Buildings 301 and 389

Transformers and other electrical equipment have been stored at Buildings 301 and 389. PCB-laden oil has been used for weed control, and spills have been reported. Soil sampling data indicate the presence of PCBs in surface soils near the former Building 389. No soil samples have been collected at Building 301. The soils around both buildings are unpaved.

Currently, workers at Buildings 301 and 389 may be exposed to site contaminants via direct soil contact and inhalation of airborne particulates. Downwind residents and recreational users of the Oakland Estuary may also be potentially exposed to airborne particulates. At this time, no contaminants other than PCBs are anticipated at these two buildings. As discussed in Section 4.1, PCBs are relatively non-mobile in the environment and bind tightly to soils. Therefore, the primary air exposure pathway for these chemicals would be inhalation of particulates, with inhalation of volatilized PCBs contributing a lesser exposure.

Under future-use conditions, access to this area may be increased, resulting in exposure to small children playing or school-aged children trespassing. If this were to occur, these populations would be identified as

potentially most exposed populations. All other future exposure pathways are the same as those identified for current conditions.

4.5.14 Cans C-2 Area

The Cans C-2 Area contains an unpaved storage yard for the variety of wastes generated at NAS Alameda. As discussed in Section 2.3, contaminants have been detected in the surface soil. Workers at the Cans C-2 area may be potentially exposed to contaminants via direct soil contact. On- and off-site workers, off-site residents and recreational users of Crab Cove may be exposed to airborne contaminants due to dust generation and volatilization from soil. Subsurface gas transport is not anticipated.

4.5.15 Station Sewer System

From 1943 to 1956, the station sewer system received untreated waste water from most of the industrial processes on base. The sewer system now receives treated waste water and storm water runoff. Explosive gases have been detected in sewer lines. Contaminants present are likely to be released via passive diffusion and bulk transport of contaminants. Potential receptors under current conditions include workers and on-site residents; potential receptors under future use conditions will be the same. Workers excavating or performing subsurface maintenance activities would be the potentially most exposed population.

4.5.16 Seaplane Lagoon

Seaplane Lagoon has received untreated waste water from NAS Alameda. Contaminants have been detected in the sediments of the lagoon. Surface water in the lagoon may also be contaminated. As discussed in Section 4.3, individuals fish in Seaplane Lagoon; no boating or swimming activities have been identified. Under current-use conditions, individuals may be indirectly exposed to surface water contaminants through the consumption of contaminated fish.

In the future, recreational boaters may have access to the lagoon and therefore, may be potentially exposed to surface water contaminants. Access to the lagoon for fishing may be increased, resulting in a larger exposed population.

4.5.17 Oakland Estuary

Contaminated sediments have been detected in the Oakland Estuary (Inner Harbor) as well as in the Oakland Outer Harbor. This area is subject to tidal flushing; therefore, surface water contamination is not anticipated in detectable quantities. Individuals may be exposed to these contaminants while engaging in recreational activities such as boating and swimming. Currently, fishing is not known to occur in this area.

Under future-use conditions, recreational use of the Oakland Estuary may increase, resulting in a larger exposed population.

4.5.18 1943-1956 Disposal Area

The 1943-1956 disposal area is now used for aviation (e.g., runways) and recreation (e.g., picnicking and jogging). The landfill is currently paved or vegetated. Surface soil contaminants have been detected in this area. Joggers and intermittent users of the picnic area may be exposed via direct soil contact or passive diffusion of volatile contaminants. Children are likely to be the most exposed population via direct contact. Landfill gas may migrate from the area to the base housing and other buildings and result in exposure of residents and workers. No information is available regarding methane gas generation at the landfill. Due to the pavement and vegetation cover, release of airborne particulates is not considered a probable current use release mechanism.

In the future, all landfill cover may be removed, which would result in larger accessible areas of contamination, thereby increasing the potentially

exposed population. Under future-use conditions, airborne contaminants may migrate by particulate transport as well as passive diffusion and bulk transport. Potential future receptors of airborne contaminants include workers, downwind on- and off-site residents, and recreational users of the Oakland Estuary.

4.5.19 West Beach Landfill

The West Beach Landfill has accepted wastes from most of the industrial operations on base. Currently, the site is fenced and access is limited to occasional workers. A wetland is located on the landfill. Surface and subsurface soil contaminants have been detected. The landfill is covered with clean fill; therefore, it is unlikely that workers will be exposed via direct contact. A more significant route of exposure is through inhalation of airborne contaminants released via passive diffusion or bulk transport of landfill gas. Workers and downwind residents as well as recreational users of Alameda Estuary may potentially be exposed to airborne contaminants.

In the future, the landfill may potentially be converted to a park or wildlife refuge incorporating the wetlands. Due to the presence of the wetlands, it is unlikely that this area will be developed in another manner. If a park were established in this area, children and adults visiting would be exposed to contaminants via direct contact.

4.5.20 Yard D-13

Hazardous waste generated by on-base activities has been stored in Yard D-13. The yard is currently paved and no contaminant release is anticipated under current conditions.

In the future, the pavement may be removed, resulting in the potential release of contaminants from surface and subsurface soils. At this time, no site history information is available to predict potential surface or subsurface soil contaminants. Future workers may be exposed via direct soil

contact. Downwind workers and residents may be exposed to contaminants via passive diffusion of volatiles or airborne particulates.

5.0 HUMAN RISK CHARACTERIZATION

According to guidelines for preparing risk assessments as part of the RI/FS process (EPA, 1986f; PRC, 1985; DHS, 1988b), the potential adverse effects on human health should be assessed, where possible, by comparing chemical concentrations found at or near the site with applicable or relevant and appropriate requirements (ARARs) or other guidance that has been developed for the protection of human health or the environment. If ARARs are available for all chemicals in all environmental media, then a comparison to ARARs constitutes the risk assessment. If not, quantitative risk estimates must be developed in addition to the comparison to ARARs. ARARs or other guidance are available only for a few of the site-related chemicals in groundwater and air at NAS Alameda; therefore, a quantitative risk characterization will be completed for air, soil, groundwater, and surface water exposure pathways in the final PHEE. A comparison with potential ARARs and other guidance and a discussion of uncertainties in this assessment are presented below.

5.1 APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS (ARARs)

5.1.1 Potential ARARs

Remedial actions selected under the Superfund Amendments and Reauthorization Act of 1986 (SARA) and the State of California Hazardous Substance Cleanup Act of 1984 must attain levels of cleanup of hazardous substances released into the environment and of control of further release which assure protection of human health and the environment. SARA specifies that any selected remedial action must achieve a level of control which at least attains requirements that are legally applicable to the hazardous substances of concern or relevant and appropriate under the circumstances of release or threatened release. Accordingly, EPA guidelines for preparing risk assessments as part of the RI/FS process (EPA, 1986f) recommend comparison of chemical concentrations found at or near a site with ARARs. Currently, the California DHS uses EPA Guidance on ARARs, with the addition of consideration of California promulgated standards and media-specific advisory levels (DHS,

1988b). Therefore, EPA's interim guidance on ARARs (EPA, 1987r) will be considered for this assessment. EPA defines ARARs as follows:

- Applicable Requirements means those cleanup standards, standards of control, and other substantive environmental protection requirements, criteria, or limitations promulgated under Federal or State law that specifically address a hazardous substance, pollutant, contaminant, remedial action, location, or other circumstance at a CERCLA site. "Applicability" implies that the remedial action or the circumstances at the site satisfy all of the jurisdictional prerequisites of a requirement. . . .

- Relevant and appropriate requirements means those cleanup standards, standards of control, and other substantive environmental protection requirements, criteria, or limitations promulgated under Federal or State law that, while not "applicable" to a hazardous substance, pollutant, contaminant, remedial action, location, or other circumstance at a CERCLA site, address problems or situations sufficiently similar to those encountered at the CERCLA site that their use is well suited to the particular site.

  The relevance and appropriateness of a requirement can be judged by comparing a number of factors, including the characteristics of the remedial action, the hazardous substances in question, or the physical circumstances of the site, with those addressed in the requirement. It is also helpful to look at the objective and origin of the requirement. For example, while RCRA regulations are not applicable to closing undisturbed hazardous waste in place, the RCRA regulation for closure by capping may be deemed relevant and appropriate.

  A requirement that is judged to be relevant and appropriate must be complied with to the same degree as if it were applicable. However, there is more discretion in this determination: it is possible for only part of a requirement to be considered relevant and appropriate, the rest being dismissed if judged not to be relevant and appropriate in a given case.

  Non-promulgated advisories or guidance documents issued by Federal or State governments do not have the status of potential ARARs. However, . . ., they may be considered in determining the necessary level of cleanup for protection of health or environment.

Only those ARARs or advisories or guidance that are ambient or chemical-specific requirements (i.e., those requirements which "set health or risk-based concentration limits or ranges in various environmental media for

specific hazardous substances, pollutants, or contaminants" (EPA, 1987r) as opposed to ARARs which are classified as action-specific or locational, are used in risk assessment. Under SARA, EPA at a minimum currently considers Maximum Contaminant Levels (MCLs) developed under the Safe Drinking Water Act, National Ambient Air Quality Standards (NAAQS), and state drinking water standards to be potential ARARs for use in risk assessment at Superfund sites. The California Department of Health Services (DHS) also considers Federal National Emission Standards for Hazardous Air Pollutants (NESHAPs) for air and California maximum contaminant levels (CMCLs) for water as ARARs.

In addition, other relevant criteria or guidance, such as EPA's Ambient Water Quality Criteria (AWQCs) and the maximum contaminant level goals (MCLGs), may be useful in assessing baseline risks or developing goals for remedial action. CDHS has also developed media-specific advisory levels known as Applied Action Levels (AALs) for use in the California Site Mitigation Decision Tree (CDHS, 1986). The AALs for air and water will also be considered as guidance in this preliminary PHEE. Potential chemical specific ARARs are identified for air and water only. No chemical specific soil criteria were available.

5.1.2 Comparison to Potential ARARs

Table 5-1 presents potential ARARs for contaminants of concern in drinking water at NAS Alameda. At this time, no current or future users of groundwater have been identified; therefore, MCLs may not be appropriate for use at this site. However, for scoping purposes, a comparison of MCLs to concentrations found in groundwater is included here.

At this time, groundwater data are only available from wells located in the West Beach Landfill, the 1943-1956 Disposal Area, Area 97, and the Cans C-2 Area. As discussed in Section 4.3.2, groundwater contaminants from different areas may impact the same well; therefore, all groundwater contaminants from differing sources on the NAS Alameda will be considered together for comparison to ARARs. Of the contaminants detected in

TABLE 5-1

Potential Applicable or Relevant and Appropriate Requirements (ARARs)
For Contaminants in Drinking Water
Naval Air Station Alameda

Chemical	Safe Drinking Water Act MCLS[a] (ug/L)	California MCLS[b] (ug/L)
Fuels		
Aviation fuel	--[c]	--
Gasoline	--	--
Oil & Grease	--	--
Volatile Organics		
Acetone	--	--
Carbon Tetrachloride	5	--
trans-1,2-Dichloroethylene	--	--
2,4-Dimethylphenol	--	--
Methyl ethyl Ketone	--	--
Methylene Chloride	--	--
Phenol	--	--
1,1,1-Trichloroethane	200	--
Tetrachloroethylene	--	2
Trichloroethylene	5	--
Vinyl Chloride	2	0.5
Monocyclic Aromatic Compounds		
Benzene	5	1
Chlorobenzene	--	--
Ethylbenzene	--	--
Toluene	--	--
Xylenes	--	--
Semivolatile Organics		
Bis(2-ethylhexyl)phthalate	--	--
PCBs	--	--
PAHS-noncarcinogenic	--	--
carcinogenic	--	--

TABLE 5-1
(continued)

Potential Applicable or Relevant and Appropriate Requirements (ARARs)
For Contaminants in Drinking Water
Naval Air Station Alameda

Chemical	Safe Drinking Water Act MCLS[a] (ug/L)	California MCLS[b] (ug/L)
Inorganics		
Antimony	--	--
Arsenic	50	--
Asbestos	--	--
Barium	1000	--
Beryllium	--	--
Cadmium	10	--
Chromium	50	--
Copper	1000[d]	--
Cyanides	--	--
Iron	300[d]	--
Lead	(5)[e]	--
Manganese	50[d]	--
Mercury	2	--
Nickel	--	--
Selenium	10	--
Silver	50	--
Thallium	--	--
Vanadium	--	--
Zinc	5000[d]	--
Pesticides		
Aldrin	--	--
Bromacil	--	--
Chlordane	--	--
Chlorvar	--	--
2,4-D	100	--
DDT	--	--
Diazinon	--	--
Diuron	--	--
Endosulfan	--	--
Endrin	0.2	--
Heptachlor	--	--
Lindane	4	--
Malathion	--	--
Monuron	--	--

TABLE 5-1
(continued)

Potential Applicable or Relevant and Appropriate Requirements (ARARs)
For Contaminants in Drinking Water
Naval Air Station Alameda

Chemical	Safe Drinking Water Act MCLS[a] (ug/L)	California MCLS[b] (ug/L)
Princep	--	--
Roundup	--	--
Warfarin	--	--

a MCLs are Maximum Contaminant Levels. MCLS are primary drinking water standards promulgated under the Safe Drinking Water Act and are based on health-related criteria and technological and economic feasibility of control.

b California MCLs established by the CDHS, Public Water Supply Branch. Only those California MCLs which are lower than federal MCLs are listed.

c -- = not promulgated; no standard available.

d Secondary MCLs. These are based on organoleptic properties and not health-based criteria.

e Proposed MCL.

groundwater, potential ARARs exist for arsenic, cadmium, chromium, copper, iron, lead, mercury, nickel, selenium, zinc, 2,4-D, endrin, lindane, trichloroethylene, and benzene. Of these contaminants, maximum concentrations of arsenic (90 μg/L), cadmium (460 μg/L), chromium (1,000 μg/L), lead (650 μg/L), mercury (3.4 μg/L), selenium (80 μg/L), trichloroethylene (291 μg/L), and benzene (9 μg/L) exceed the potential ARARs presented in Table 5-1. Benzene and trichloroethylene were found in highest concentrations near the 1943-1956 Disposal Area; all other maximum groundwater concentrations were found in the West Beach Landfill. The DHS has no ARARs for total oil and grease levels.

In addition to ARARs, EPA and DHS recommend the use of other guidance for sites undergoing cleanup. Table 5-2 presents other guidance values for groundwater contaminants. In addition to those contaminants which exceed their respective ARARs, contaminants which exceed these guidance values may also contribute to human health risk from a site. Toluene (235 μg/L), noncarcinogenic PAHs (120 μg/L), and nickel (400 μg/L) exceed their respective California AALs for water; trans-1,2-dichloroethylene (957 μg/L) exceeds the federal maximum contaminant level goal (MCLG) of 70 μg/L.

Table 5-3 presents potential ARARs and guidance values for potential airborne contaminants originating from NAS Alameda. Currently, no data are available to compare to these ARARs.

5.2 RISK CHARACTERIZATION

For those contaminants where ARARs are not available, a quantitative risk assessment is performed to evaluate media-specific risks. For NAS Alameda, no surface water ARARs were identified and no soil ARARs are available.

To quantitatively assess the potential risks to human health associated with the current-use and future-use exposure scenarios considered in this assessment, the concentrations of chemicals in relevant environmental media at

TABLE 5-2

Other Relevant Criteria and Guidance
for Waterborne Contaminants
Naval Air Station Alameda

Chemical	Safe Drinking Water Act MCLGs[a] (ug/L)	California Applied Action Levels[b] (ug/L)
Fuels		
Aviation fuel	-- [c]	--
Gasoline	--	--
Oil & Grease	--	--
Volatile Organics		
Acetone	--	--
Carbon Tetrachloride	0	--
trans-1,2-Dichloroethylene	70	--
2,4-Dimethylphenol	--	--
Methyl ethyl ketone	--	1770
Methylene Chloride[e]	--	--
Phenol	--	--
1,1,1-Trichlorethane	200	200
Tetrachloroethylene	--	--
Trichloroethylene	(0)[d]	7
Vinyl Chloride	0	--
Monocyclic Aromatic Compounds		
Benzene	0	0.7
Chlorobenzene	60	--
Ethylbenzene	680	680
Toluene	2000	100
Xylenes	440	620
Semivolatile Organics		
Bis(2-ethylhexyl)phthalate	--	--
PCBs	0	--
PAHs-noncarcinogenic	--	19 [e]
carcinogenic[f]	--	2.3×10^{-5}

TABLE 5-2
(continued)

Other Relevant Criteria and Guidance
for Waterborne Contaminants
Naval Air Station Alameda

Chemical	Safe Drinking Water Act MCLGs[a] (ug/L)	California Applied Action Levels[b] (ug/L)
Inorganics		
Antimony	--	--
Arsenic	50	--
Asbestos	7.1×10^6 g	--
Barium	1500	--
Beryllium	--	--
Cadmium	5	--
Chromium	120	--
Copper	1300	--
Cyanides	--	--
Iron	--	--
Lead	0	--
Manganese	--	--
Mercury	3	2
Nickel	--	400
Selenium	45	--
Silver	--	--
Thallium	--	--
Vanadium	--	--
Zinc	--	7500
Pesticides		
Aldrin	--	--
Bromacil	--	--
Chlordane	0	--
Chlorvar	--	--
2,4-D	70	35
DDT	--	--
Diazinon	--	--
Diuron	--	--
Endosulfan	--	--
Endrin	--	--
Heptachlor	0	--
Lindane	0.2	--
Malathion	--	--
Monuron	--	--

TABLE 5-2
(continued)

Other Relevant Criteria and Guidance
for Waterborne Contaminants
Naval Air Station Alameda

Chemical	Safe Drinking Water Act MCLGs[a] (ug/L)	California Applied Action Levels[b] (ug/L)
Princep	--	--
Roundup	--	--
Warfarin	--	--

a MCLGs are Maximum Contaminant Level Goals promulgated under the Safe drinking Water Act using health-based criteria.

b California AALs are developed for use in CDHS Site Mitigation Decision Tree.

c No criteria available.

d Proposed MCLG.

e AAL for noncarcinogenic PAHs is for one or more noncarcinogenic PAHs.

f Carcinogenic PAHS are represented by benzo(a)pyrene.

g Asbestos concentrations in fibers/liter.

TABLE 5-3

Potential Applicable or Relevant and Appropriate Requirements
and Other Guidance Values
for Airborne Contaminants
Naval Air Station Alameda

Chemical [d]	ARARs		Other Guidance
	AAQS [a] (ug/m^3)	NESHAPs [b] (ug/m^3)	California AALs [c] (ug/m^3)
Organic Contaminants			
Benzene	--	source[e]	3.2
2,4-D	--	--	3.5
Ethylbenzene	--	--	140
Methyl Ethyl Ketone	--	--	250
PAHs - noncarcinogenic[f]	--	--	1.9
carcinogenic[g]			8.7×10^{-6}
Toluene	--	--	200
1,1,1-Trichloroethane	--	--	310
Xylene	--	--	100
Vinyl Chloride	26 (CA)	source	--
Inorganic Contaminants			
Arsenic	--	source	--
Asbestos	--	no visible discharge	--
Beryllium	--	0.01[h]	--
Lead	1.5(CA/EPA) [h,i]	--	--
Mercury	--	source	0.007
Nickel	--	--	0.1
Zinc	--	--	0.75
Particulate Matter	50 (CA) [j] 30 (CA) [k]	--	--
Hydrocarbons (non-methane)	160 (EPA) [l]	--	--

a AAQS - Ambient Air Quality Standards both federal and state standards. EPA = federal, CA = state.
b NESHAPs - National Emission Standards for Hazardous Air Pollutants
c AALs - Applied Action Levels
d Only those chemicals with air standards are included
e Source emission standard for manufacturing. Benzene and vinyl chloride are regulated to prevent fugitive emissions.
f For all non-carcinogenic PAHs, except naphthalene (1.8 ug/m^3)
g Carcinogenic PAHs represented by benzo(a)pyrene.
h 30-day average.
i 90-day average.
j 24-hour average.
k 1-year average.
l 3-hour average.

points of potential exposure (exposure point concentrations) are developed and then are converted to chronic daily intakes (CDIs). CDIs are expressed as the amount of a substance taken into the body per unit body weight per unit time, or mg/kg/day. A CDI is averaged over a lifetime for carcinogens, and over the exposure period for noncarcinogens (EPA, 1986f; 1986i). For potential carcinogens, the upper-bound, 95% confidence interval excess lifetime cancer risks are obtained by multiplying the chronic daily intake of the contaminant under consideration by its cancer potency factor. Overall cancer risk is then determined by summing all chemical specific cancer risks.

Potential risks for noncarcinogens are presented as the ratio of the chronic daily intake of a chemical to its Reference Dose (RfD), presented in Section 3. The sum of the CDI:RfD ratios of all noncarcinogenic chemicals under consideration is called the Hazard Index. The Hazard Index is useful as a screening reference point for gauging the potential effects of environmental exposures to complex mixtures. In general, Hazard Indices which are less than one are not likely to be associated with any health risks, and are therefore less likely to be of regulatory concern than Hazard Indices greater than one. A conclusion should not be categorically drawn, however, that all Hazard Indices less than one are "acceptable" or that Hazard Indices of greater than one are "unacceptable." This is a consequence of the greater uncertainty inherent in estimates of the RfD and CDI, in addition to the fact that the uncertainties associated with the individual terms in the Hazard Index calculation are additive.

In accordance with EPA's guidelines for evaluating the potential toxicity of complex mixtures (EPA, 1986k), it is assumed that the toxic effects of the site-related chemicals would be additive. Thus, for each exposure scenario, lifetime excess cancer risks and are summed to indicate the potential risks associated with mixtures of potential carcinogens. CDI:RfD ratios are similarly summed to indicate potential risks associated with mixtures of noncarcinogens. If the initial screening effort results in a Hazard Index equal to, or greater than, 1.0, then the individual chemicals are to be re-grouped according to their toxic endpoints and the summed CDI:RfD

ratios reevaluated (EPA, 1986k). In the absence of specific information on the toxicity of the mixture to be assessed or on similar mixtures, EPA guidelines generally recommend assuming that the effects of different components of the mixtures are additive. Synergistic or antagonistic interactions may be taken into account if there is specific information on particular combinations of chemicals. In this risk assessment, it was assumed that the potential effects of the site-related chemicals would be additive.

As discussed in Sections 4.4 and 4.5, individuals currently on the NAS Alameda site are most likely to be exposed to contaminants present in surface soils, surface water, and subsurface gases. In this section, potential human health risks are evaluated using the data presented in Section 2, along with the exposure pathway assessment presented in Section 4. Due to the limited sampling data available, this discussion will be qualitative in nature and, thus, cannot completely address all of the areas of suspected contamination.

5.2.1 Soil Pathways

As discussed in Section 4.4, individuals working or engaged in recreational activities in unpaved areas where surface soil contamination is present may be at risk to exposure to chemicals via direct soil contact. Direct contact with contaminated soil can lead to exposure via inadvertent or intentional soil ingestion via hand-to-mouth contact, deposition in the upper respiratory tract of inhaled particles which are subsequently swallowed, or dermal absorption. Inorganic contaminants, with the exception of cyanide, tend to be poorly absorbed through intact skin, and dermal absorption is expected to be insignificant relative to gastrointestinal absorption for these chemicals. The lipophilic organic chemicals (e.g., benzene, DDT) are absorbed through the skin much more easily than inorganics, and for these chemicals both dermal absorption and ingestion routes must be considered.

Individuals working on-site are anticipated to have the highest incidence of contact with contaminated soils. Previous investigations have indicated the presence of contaminants in the crawl space below Building 360.

Although some of the contaminated soil was subsequently removed, additional contamination is suspected and may pose an occupational risk to workers in that building. Other sites (e.g., Cans C-2 Area) have known or suspected soil contamination but do not have as high a probable frequency of contact. Therefore, these areas may not pose a potential health risk at this time. Since the picnic area is used infrequently (if at all), human health risks to users of this area are anticipated to be minor. If this area is returned to extensive active use, then it may pose a significant risk to picnickers, especially young children.

In order to quantitatively assess potential human health risks posed by direct soil contact at NAS Alameda, additional data are required. In areas where direct contact may occur, data from surface soils (0-6 inches) are needed to identify the chemicals present. The types of contaminants will influence whether contaminants are likely to be absorbed dermally through the skin or ingested due to hand to mouth contact. Information is also necessary on the potential receptor populations. For example, if workers are anticipated to be the exposed population, the duration of exposure and the rate of ingestion would be significantly smaller than if young children were to be the anticipated exposed population. It is anticipated that on-site workers are the only exposed receptor population that may receive any significant exposure to surface soil contaminants.

5.2.2 Surface Water Pathways

As discussed in Sections 4.2 and 4.4, fish in contact with contaminated surface water and sediments may absorb these contaminants and store them in fatty tissue. Lipophilic organic contaminants are likely to partition from water into fatty tissue, thus resulting in bioconcentration of contaminants. Concentrations of contaminants in fish can be estimated using bioconcentration factors and surface water or sediment concentrations. Exposures to individuals consuming contaminated fish can be estimated using fish consumption averages available from USDA. Inorganic contaminants with the exception of mercury, are not expected to bioconcentrate.

Storm water and surface water runoff from NAS Alameda are discharged into the Seaplane Lagoon. Also, hydrocarbon vapors have been detected in the station sewer system which discharges to the lagoon. All these discharges may contribute to contamination in the Lagoon, resulting in contaminant uptake by fish in this area. Individuals fishing in Seaplane Lagoon may be exposed to contaminants present in surface water, and sediments and subsequently absorbed by fish in the lagoon. The rate of fish consumption by these individuals is unknown, as is the concentration of contaminants present in fish in this area. However, fish in this area have been reported to smell "oily". As more data are available, the risk to individuals fishing in this area will be quantified. At this time, it appears that consumption of fish from the Seaplane Lagoon may result in significant human health risk.

In order to evaluate human health risk due to contaminants in surface waters at NAS Alameda, surface water, and sediment analytical data are necessary. With this information, chemicals likely to bioconcentrate can be identified; biota samples can then be analyzed for these contaminants. Additional demographic data identifying the frequency of fishing at NAS Alameda will aid in the generation of site-specific risk estimates.

An additional pathway of potential human exposure is via the inhalation of volatile contaminants being released from surface waters in the lagoon. In general, although volume dilution in the atmosphere would tend to greatly reduce the actual airborne concentrations of any volatiles, individuals near the discharge points of the sewer system could potentially be exposed to higher airborne concentrations. Estimation of the rate of sewer discharge to the lagoon as well as information on the contaminants present in this discharge are necessary to evaluate this pathway.

5.2.3 Air Exposure Pathways

As discussed in Section 4.2, volatile organic compounds may be transported to locations distant from the site by vapor phase transport and

subsequent volatilization. Contaminants can migrate through permeable lithologic zones and through manmade conduits, such as sewers and underground lines. Individuals working near underground lines or living in houses with basements may potentially be exposed to subsurface contaminants.

Subsurface gas vapors have been detected in underground ducts near Area 97. In some areas, concentrations of these vapors have exceeded the explosive range of these contaminants. The contaminants present are anticipated to be volatile constituents of AVGAS (e.g., benzene, toluene, and xylene). Workers in subsurface areas may be exposed to these chemicals. Depending upon the extent of lateral migration, the potential for exposure likewise exists for off-site residents with basements. No monitoring has been performed in residential homes. During the RI, gas wells will be installed to determine whether AVGAS contaminants are migrating off-site. Any potential risk to off-site residents will be quantified when the data are available. These vapors may also represent an explosion hazard to individuals working near underground lines.

In order to quantify exposures to subsurface gases, information is needed regarding subsurface conditions including contaminants present, potential permeable units and manmade conduits, and potential receptors. Those organic chemicals with sufficient vapor pressures, as discussed in Section 4.1, may be transported as a gas through permeable zones. The direction and rate of flow of subsurface vapors is necessary to identify receptor populations. Information on the potential receptor population is necessary to appropriately quantify risks. For example, if residents of houses with basements are identified as the most likely receptor population, then the duration and frequency of exposure may be longer than for occasional subsurface workers. Also, residential populations generally contain more sensitive subpopulations, such as children and the elderly. If an off-site receptor population is identified, gas monitoring or modeling may be necessary to quantify exposure point concentrations for potential receptors.

Additional environmental release mechanisms for contaminants have been identified in Section 4.2: generation of airborne particulates and passive diffusion of volatiles from soils. In order to quantify risks from air contaminants, information is needed regarding average wind speed and direction as well as the types of contaminants detected. Meteorological information will be used to identify potential downwind receptors. The type of contaminant will influence the potential environmental release mechanism. Volatile organics are more likely to be released via passive diffusion, whereas inorganics and semivolatile organics (e.g., PAHs, DDT) are more likely to be released via particulate transport. Workers in areas with unpaved contaminated soils would be most at risk to these airborne contaminants due to their proximity to the emission source.

As discussed previously, limited data were available for this assessment and therefore, human health risks could not be quantified. Limitations in the current data which hinder the evaluation of the exposure pathways presented in Sections 4.4 and 4.5 were identified here and will be evaluated in detail in Section 7. Those sites that appear to pose the most significant health risks due to the presence of contamination and a completed exposure route are the Seaplane Lagoon, Area 97, and Building 360.

5.3 UNCERTAINTIES IN THE RISK ASSESSMENT FOR HUMAN HEALTH

The procedures and inputs used to assess potential human health risks in this evaluation are subject to a number of uncertainties. One of the objectives of this preliminary PHEE is to identify data deficiencies and uncertainties in order to generate a high quality final PHEE. In general, there are six main sources of uncertainty:

- environmental chemistry sampling and analysis,
- environmental parameter measurements,
- fate and transport modeling,
- toxicological data and dose-response extrapolations,

- assumptions concerning exposure scenarios, and
- errors through combinations of the above.

These general sources of uncertainty as they pertain to this assessment are discussed below.

Environmental chemistry sampling and analysis error can stem from error inherent in the procedures, from a failure to take an adequate number of samples to arrive at sufficient areal resolution, from mistakes on the part of the sampler, or from the heterogeneity of the matrix being sampled. In an attempt to reduce the influence of this on the data, Canonie is proposing to collect samples from all environmental media and analyze them for all suspected contaminants. Quality assurance and quality control procedures will be followed to ensure sample integrity.

Absence of environmental parameter measurements contributes to uncertainty. Estimates must therefore be made based on literature values, regression equations, extrapolations, and best professional judgements. For example, in the final PHEE, estimates of uptake of contaminants by fish in Seaplane Lagoon may over- or underestimate actual conditions.

In the absence of reliable field data detailing the concentration of contaminants at exposure points, fate and transport modeling is conducted to estimate these concentrations. Actual concentrations of contaminants at exposure points may be quite different from predicted concentrations. For example, airborne migration of contaminants from NAS Alameda study areas may be predicted in the final EA using models developed for other sites.

The results of animal studies are often used to predict the potential health effects of a chemical in humans. Extrapolation of toxicological data from animal tests is one of the largest sources of uncertainty in risk assessment. There may be important but unidentified differences in uptake, metabolism, and distribution of chemicals in the body between the test species and man.

Typically, animals are administered high doses of a chemical in a standard diet. Humans, on the other hand, may be exposed to much lower doses in a highly variable diet. In these studies, animals, usually laboratory rodents, are exposed daily to the chemical agent for various periods of time up to their 2-year lifetime. Humans have a 70-year lifetime and may be exposed either intermittently or regularly for an exposure period ranging from months to a full lifetime. Because of these differences, it is not surprising that extrapolation error is a large source of uncertainty in risk assessment. Even if studies in humans are available, uncertainties can be large because the diet, activity patterns, exposure duration and frequency, and individual susceptibility may not be the same in the study populations as in the individuals exposed to environmental concentrations.

Exposure estimation is another potentially large source of error in risk assessment. The potential risks calculated in the final PHEE will depend largely on the exposure pathways selected for evaluation. Selection of exposure pathways is a process, often based on best professional judgement, which attempts to identify the most probable and potentially harmful exposure scenarios. In a risk assessment, it is possible that risks are not calculated for all of the exposure pathways.

Uncertainties from different sources will be compounded in the risk assessment. For example, errors that were introduced while sampling may be compounded by use of this potentially erroneous data in modeling efforts.

6.0 PRELIMINARY ENVIRONMENTAL ASSESSMENT

This section assesses potential impacts to aquatic and terrestrial environments which may result from exposure to the chemical contaminants identified at NAS Alameda. The approaches used in this assessment roughly parallel those used in human health risk assessment in that potentially exposed populations (receptors) are identified, and then information on exposure and toxicity is combined to derive estimates of risk.

This section is organized around this basic assessment procedure. In Section 6.1, potential environmental receptors are identified. Potential exposure pathways are evaluated in Section 6.2, and available toxicity data are summarized in Section 6.3. Finally, in Section 6.4, potential risks are qualitatively evaluated. Discussions of risks are limited primarily to the population (species) level because data on community and ecosystem level responses to environmental pollutants generally are lacking. However, where possible, the implications of population level impacts on the community or ecosystem are discussed.

6.1 RECEPTOR CHARACTERIZATION[1]

In this section the plant and animal species which occur at NAS Alameda and the surrounding area are identified and indicator species or species groups are selected for further evaluation. Species representative of the site and surrounding area are listed in Appendix B and were identified based on information obtained during a site visit, from contacts with State and Federal government personnel, and from previously published reports (EAE/WESTDIV, 1974; E&E/WESTDIV, 1983; Collins, 1987; Bailey, 1987).

---

[1]Common species names are used in this section. Scientific names are presented along with the common names in Appendix B.

6.1.1 Terrestrial Receptors

NAS Alameda is located on Alameda Island in the southern portion of San Francisco Bay. Wildlife habitat at NAS Alameda is limited primarily to the western half of the site; the eastern half is heavily developed with office buildings and industrial facilities. Habitat in the western half is characterized principally by upland areas vegetated by annual grasses and low-lying shrubs.

Two brackish water wetlands also occur in the western portion of NAS Alameda (COE, 1985). A two to three acre wetland occurs in the southwest portion of the West Beach Landfill and a one to two acre wetland occurs west of the Seaplane Lagoon. Wetland plant species in both areas include pickleweed, brass buttons, and bulrushes (COE, 1985). Vegetation in the landfill wetland is sparse and some of the area in the northwestern portion of this wetland has been disturbed and appears to have been partially filled. Small ponded areas occur in the southeastern portions of the landfill wetland. The Seaplane Lagoon wetland is seasonally wet and was dry at the time of a site visit in October, 1988.

The West Beach Landfill area, and to a more limited extent the 1943-1956 Disposal Area, provides some good wildlife habitat. The heavy vegetation of the landfill likely provides good cover for small mammals as well as serving as a food source for small mammals and some land-foraging birds. The presence of small mammals also provides a food resource for hawks and other raptors. The wetland area is also likely to be good habitat for dabbling ducks and shorebirds. In addition to the upland and wetland habitat areas, the Bay waters adjacent to NAS Alameda provide foraging habitat for a variety of birds.

Mammal species known or expected to occur in the landfill area are black-tailed jackrabbits, voles, house mice, western harvest mice, western ground squirrels, raccoons, and feral cats. Bird species known or expected to use the landfill and surrounding area for foraging include killdeer,

loggerhead shrike, black-shouldered kite, red-tailed hawk, Northern harrier, American kestrel, and peregrin falcon. Burrowing owls also may occur in the landfill (Kelly, 1988).

Bird species known or expected to use the wetland areas include ducks, coots, plovers, avocets, stilts, willets, sandpipers, gulls, herons, egrets and terns. A large colony of Caspian terns is known to use the landfill wetland (Worthing, 1988).

Bird species foraging in the Bay surrounding the site include Forster's tern, Caspian tern, elegant tern, double-crested cormorant, California, herring, ring-billed, glaucous-winged, and Bonapart's gulls, western grebe, pied-billed grebe, common goldeneye, white-winged and surf scoters, greater and lesser scaups, canvasback, bufflehead, ruddy duck, and red-throated loon. A variety of shore birds forage on the riprap that surrounds the majority of the western portion of NAS Alameda. A large colony of western gulls nests on the breakwater island south of the Seaplane Lagoon, and brown pelicans use this area as a roost in the late summer and fall (Worthing, 1988). Both species likely forage in the surrounding waters. In addition, California least terns, which have a breeding colony at NAS Alameda near the runways east of the landfill, use the waters adjacent and south of NAS Alameda for foraging (Bailey, 1987). Foraging is greatest near the breakwater area, in the Seaplane Lagoon, and in the offshore areas directly south of the landfill (Bailey, 1987).

6.1.2 Aquatic Species

The estuarine waters adjacent to NAS Alameda support a variety of aquatic species (EAE/WESTDIV, 1974). Benthic species known to occur in the waters adjacent to NAS Alameda include polychaete worms, white acorn barnacle, mud crab, amphipods, and bryozoans. A number of molluscs also occurs in the estuarine sediments, including mud snail, bay mussel, California mussel, native oyster, littleneck clam, Japanese littleneck clam, soft-shelled clam, and bent-nosed clam. Fish of the area include Pacific herring, northern

anchovy, jack smelt, top smelt, starry flounder, shad, surf perch, striped bass, chinook and coho salmon, white croaker, and yellowfin goby.

6.1.3 Endangered and Threatened Species

Two Federal and State endangered species occur at NAS Alameda. These are the California least tern and brown pelican (Kelly, 1988). The terns breed at NAS Alameda, and both species forage in the waters south of NAS Alameda. In addition to these species, several State species of special concern occur at the site. These are double-crested cormorants, northern harrier, and elegant tern (Kelly, 1988). Burrowing owls, also a State species of special concern, may occur at NAS Alameda in the landfill area.

6.1.4 Selection of Indicator Species or Species Groups for Further Evaluation

As the previous discussion indicates, NAS Alameda and the surrounding area supports a variety of plant and animal species. Because of this diversity, it is not feasible to assess impacts to every species potentially affected. A common approach to this problem in ecological evaluations is to select "indicator" species or species groups for detailed evaluation and to assume that impacts to these species are representative of potential impacts on other species at the site. The selection of indicator species or groups is based on several factors, including the potential for exposure, the availability of toxicity data, and the "health" of the potentially impacted populations (e.g., threatened or endangered species). Each of these factors was considered in the selection of indicators for the preliminary environmental assessment of NAS Alameda.

The selection of indicator species of plants at NAS Alameda is driven primarily by the availability of toxicity data since no differences in exposure potential exist between the species and no endangered or threatened plants are expected to occur at the site. Unfortunately, toxicity information specific to the plant species that occur at the site is not available.

Therefore, an indicator species is not selected, but rather, plants as a group are selected as indicators.

For terrestrial wildlife species, the potential for exposure is an important factor in the selection of indicator species for this assessment. Mammal and bird indicator species are selected based on feeding habits, because exposure to the chemical contaminants via food is considered to be an important route of exposure for wildlife at NAS Alameda. Jackrabbits are chosen as an indicator species representing herbivorous mammals of the site. Killdeer are selected as an indicator species representative of land-foraging birds, and plovers are selected as a species representative of birds that feed in the wetland and shore areas. Although many water-foraging birds occur at NAS Alameda, the California least tern and brown pelican are selected as indicators of this group because they are also classified as Federal and State endangered species.

The selection for aquatic indicator species is driven by the availability of toxicity data. Toxicity criteria are available for aquatic life as a whole and therefore, no particular species is selected as an indicator, but rather all aquatic life will be evaluated.

6.2 POTENTIAL EXPOSURE PATHWAYS

In this section, the pathways by which plants and wildlife may be exposed to the chemical contaminants in the soil, groundwater, surface water, and sediments at NAS Alameda are identified. Monitoring data needed to quantify exposure also are identified. The following discussion is organized by receptor type.

6.2.1 Terrestrial Plants

Terrestrial plants may be exposed to chemical contaminants in the soil. Exposure is generally limited to those chemicals that occur within the root zone, and exposure results from plant uptake through the roots. The depth of

the root zone varies with the species, but for grasses and small shrubs, such as those in the West Beach Landfill and the 1943-1956 Disposal Area, the root zone can be expected to be limited to the top one to two feet of soil.

No data are available, however, on the chemical contaminants within the shallow soil at NAS Alameda. Soil data are limited to analyses of soil borings from the 1943-1956 Disposal Area taken at a depth of 6 to 6.5 feet. Plant exposure to these contaminants in the deeper soils is unlikely and therefore is not quantified in this preliminary assessment. Data from the shallow (0 to 1 foot) soils of 1943-1956 Disposal Area and the West Beach Landfill are needed to evaluate potential impacts to plants at NAS Alameda.

6.2.2 Terrestrial Wildlife

Terrestrial wildlife may be exposed to chemical contaminants in the soil, sediment, and surface water at NAS Alameda. Exposure may occur directly by ingestion of or direct contact with contaminated media or indirectly by ingestion of food (e.g., plants, invertebrates, fish) that has accumulated chemicals from contaminated media. In many cases, however, adequate data are not available to estimate exposure via all of these pathways. For example, although it is known that mammals can ingest soil contaminants while preening and birds could ingest sediments while foraging on benthic species, it is not known how much soil or sediment is actually ingested during these activities. Similarly, the amount of chemical that is dermally absorbed following direct contact with contaminated soil, sediment, or surface water by mammals or birds is not known. Therefore, direct ingestion of soil or sediment and dermal absorption of contaminants will not be addressed further here.

Potential pathways by which terrestrial wildlife may be exposed at NAS Alameda and for which exposure assessment data exist are ingestion of surface water and ingestion of food that has accumulated contaminants from surface water, sediment, or soil. However, no surface water data are available from the Seaplane Lagoon, Bay waters, and the on-site wetlands, and no sediment data are available from the wetland areas. Further, no data are available

from the shallow soils of the 1943-1956 Disposal Area and the West Beach Landfill. Therefore, exposure to wildlife in these areas cannot be quantified in this preliminary assessment, although exposure via all of these pathways is potentially important at NAS Alameda. Below, we discuss receptor-specific exposure pathways for terrestrial wildlife at NAS Alameda and present approaches for quantifying exposure if appropriate monitoring data were available.

6.2.2.1 Black-tailed Jackrabbits (Herbivorous Mammals)

Jackrabbits are herbivorous, ground-dwelling mammals. Based on the habitat characteristics of NAS Alameda, jackrabbits would be expected to live and forage in the West Beach Landfill area. In addition to dermal absorption and direct soil ingestion (discussed above), exposure can occur by ingestion of contaminants in surface water used as drinking water, and by ingestion of food (vegetation) that has accumulated contaminants from the soil. Each of these pathways is potentially complete.

If surface water data were available, drinking water exposures could be quantified using daily water ingestion rates, the body weight of jackrabbits, and the concentration of the chemical in the drinking (surface) water.

Shallow soil data from the landfill are needed to characterize exposure of jackrabbits via plant consumption. Exposures via food could be evaluated by estimating the uptake of contaminants from soil by plants and the quantity of food (plants) ingested daily by a jackrabbit. Estimates of plant uptake can be generated using chemical concentration data from shallow soils (0 to 1 foot), which are representative of contaminants potentially accumulated by plants, and a vegetation:soil concentration ratio for the chemical (mg/kg plant per mg/kg in soil). Of the chemicals detected in the deep soil samples of the disposal area (and potentially present in the landfill surface soils), vegetation:soil concentration factors are available for arsenic, cadmium, chromium, lead, mercury, nickel (Clement Associates, 1988), and PAHs (Edwards, 1983). Therefore these chemicals are selected for further evaluation in this

preliminary assessment of potential impacts on jackrabbits and other herbivorous mammals at NAS Alameda.

6.2.2.2 Killdeer (Land-foraging Birds)

Killdeer generally live in open fields, heavily grazed meadows, or other dry uplands with closely cropped or sparse vegetation (USDA, 1987). Based on these habitat requirements, killdeer at NAS Alameda would be expected to occur in the 1943-1956 Disposal Area and in the less vegetated areas of the West Beach Landfill. As was the case for the jackrabbit, exposure to chemicals can occur by ingestion of contaminants in surface water used as drinking water and by ingestion of food that has accumulated contaminants from the soil.

Again, were surface water data available, drinking water exposures could be estimated using assumptions regarding body weight and daily water ingestion rates. For dietary exposures, the concentration of chemicals in the food of killdeer must be estimated. Shallow soil data from the 1943-1956 Disposal Area and the West Beach Landfill are most appropriate to evaluate this pathway. The primary food items of killdeer are worms, insects, spiders, some aquatic invertebrates, and weed seeds (USDA, 1987). Killdeer food exposures can be estimated by assuming that some of the contaminants in the soil are concentrated in the ground-dwelling prey of killdeer. Of the chemicals detected in the deep soil samples of the 1943-1956 Disposal Area (and potentially present in the surface soils of the disposal area and the landfill soils), data are available which relate the concentration of chromium, copper, lead, cadmium, and zinc in soil to their concentration in earthworms (Diercxsens et al., 1985). Therefore these chemicals are selected for further evaluation in this preliminary assessment of potential impacts on killdeer and other land-foraging birds.

6.2.2.3 Plovers (Wetland and Shore Birds)

Plovers and other wetland and shore birds may be exposed to chemical contaminants in the sediments and surface water of the wetland areas at NAS

Alameda. The most likely exposure pathways are ingestion of surface water and ingestion of invertebrates that have accumulated contaminants from the sediments and surface water. Because surface water and sediment data are not available, exposure of plovers and similar species cannot be evaluated. Were data available, drinking water exposures could be estimated by making assumptions regarding drinking water intakes and body weights. Food exposures could be estimated by first estimating the concentration of chemicals in the plover's food using water to organism bioconcentration factors and then estimating daily food intake.

6.2.2.4 California Least Terns and Brown Pelicans (Bay-foraging Birds)

The most probable pathway by which terns and pelicans may be exposed to chemical contaminants at NAS Alameda is ingestion of surface water and ingestion of fish that have accumulated the contaminants from surface water. Again however, this pathway cannot be quantified at the present time because of lack of data. Were data available, exposures could be estimated in a manner similar to that described above for plovers (i.e., estimate concentration in food and daily food intake as well as drinking water ingestion rates).

6.2.3 Aquatic Life

Aquatic life may be exposed to chemical contaminants in sediment and surface water. No surface water monitoring data are available. It is believed, however, that all groundwater at NAS Alameda eventually discharges into the surrounding surface waters, and thus groundwater is a potential source of surface water contamination. Surface water exposure point concentrations cannot be estimated based on the available groundwater data because the characteristics of groundwater flow, the concentration of chemicals in the groundwater at the point of surface discharge, and chemical dispersion in the surrounding surface waters is not known or cannot yet be estimated. Nevertheless, in this preliminary assessment, we will conservatively assume that the concentrations in groundwater represent the

concentrations of the contaminants at the point where they enter surface water. Exposure point concentrations are therefore the groundwater concentrations reported in Section 2.3 for the West Beach Landfill, 1943-1956 Disposal Area, Area 97, and the Cans C-2 Area. Aquatic organisms may be exposed to these contaminants at the point of groundwater discharge. Exposure will be greatest for benthic species living at the discharge point; mobile species (e.g., fish) and benthic species living further from the discharge point would be exposed to more dilute concentrations.

Sediment data are available for the Seaplane Lagoon and the Oakland Inner Harbor. Sediment toxicity criteria have not been developed for the inorganic chemicals detected in these sediments. Therefore, risks are assessed by estimating the concentration of these compounds in the sediment interstitial water and comparing these concentrations to the ambient water quality criterion. The interstitial water, in this instance, is the exposure point rather than the sediments themselves. Under this approach, it is assumed that the toxicity and accumulation of the contaminants by benthic organisms is correlated to the interstitial concentration of the contaminant and not directly to the total concentration of the contaminant in the sediment. Also, it is assumed that interstitial water concentrations of the contaminants are controlled by partitioning between the sediment and the interstitial water and that this can be approximated by the following equation:

$$C_w = C_s/K_d$$

where

C_w = Chemical concentration in interstitial water (mg/l),

C_s = Chemical concentration in sediments (mg/kg),

K_d = Partitioning coefficient between sediment and water.

This approach however ignores site-specific sediment parameters which control the partitioning of inorganic chemicals, such as amorphous hydrous oxides of iron and manganese and reactive particulate organic carbon content of the sediment. In the absence of such site specific data for NAS Alameda

and surrounding area, K_ds derived from the literature are used in this assessment. An example of measured sediment concentrations and estimated pore water concentrations are presented in Table 6-1.

6.3 TOXICITY OF CHEMICAL CONTAMINANTS AT NAS ALAMEDA

In this section, the toxicity of selected chemicals that occur in the soil, sediment, and groundwater of NAS Alameda is discussed and critical toxicity values are identified for use in the assessment of potential environmental impacts. The discussion of toxicity is organized around the previously identified receptors (plants, jackrabbits, birds, and aquatic life). Based on the available monitoring data and the preliminary exposure pathway analysis, toxicity data for jackrabbits (mammals) are presented only for arsenic, cadmium, chromium, lead, mercury, nickel, and PAHs. Toxicity data for killdeer (birds) are presented only for chromium, copper, lead, and zinc. Toxicity data for plants are presented for those chemicals detected in the soils of the disposal area for which toxicity information is available. Toxicity data for which aquatic life are presented for those chemicals in groundwater and sediments for aquatic toxicity criteria have been developed.

6.3.1 Toxicity to Terrestrial Plants

Toxicity criteria for the protection of terrestrial plants have not been established. In this preliminary assessment, soil contamination concentrations associated with no-observable-adverse-effect levels (NOAELs) are used to establish acceptable levels for plants. If NOAELs are not available, then lowest-observable-adverse-effect levels (LOAELs) are used. Of the chemicals detected in soil from the 1943-1957 Disposal Area, phytotoxicity data are available for arsenic, cadmium, chromium, copper, and lead. Toxicity criteria are summarized in Table 6-2.

TABLE 6-1

SEDIMENT CONCENTRATIONS AND ESTIMATED PORE WATER CONCENTRATIONS FOR THE SEA PLANE LAGOON AND THE OAKLAND INNER HARBOR

Chemical	Kd (b) (ml/g)	Sediment Concentration (mg/kg) (a)		Estimated Pore Water Concentration (mg/l)	
		Sea Plane Lagoon	Oakland Inner Harbor	Sea Plane Lagoon	Oakland Inner Harbor
Arsenic	5	5.7 (c)	6	1.1	1.2
Barium	60	25	ND	0.42	ND
Cadmium	6.5	1.5	0.42	0.23	0.065
Chromium	20 (d)	50	66	2.5	3.3
Copper	35	33	47	0.94	1.3
Lead	900	31	22	0.034	0.024
Mercury	10	0.18	0.39	0.018	0.039
Nickel	40	34	61	0.85	1.5
Selenium	5	9	ND	1.8	ND
Silver	45	ND	0.1	ND	0.0022
Thallium	1500	6.9 (c)	ND	0.0046	ND
Zinc	40	71	106	1.8	2.7

(a) Value is the geometric mean concentration unless otherwise noted.

(b) Values reported in ICF (1986). These values have been derived for soil and have not been validated for sediments but are used here in lieu of more appropriate data

(c) Maximum value.

(d) Value for Cr+6.

ND = Not detected in sediments from this area.

TABLE 6-2

PLANT TOXICITY VALUES FOR THE PRELIMINARY ENVIRONMENTAL ASSESSMENT

Chemical	Critical Toxicity Value (mg/kg soil)	Basis for Toxicity Value	Reference
Arsenic	15	LOAEL	Kabata-Pendias and Pendias (1984)
Cadmium	3	LOAEL	Kabata-Pendias and Pendias (1984)
Chromium	0.01	LOAEL	NAS (1974)
Copper	60	LOAEL	Kabata-Pendias and Pendias (1984)
Lead	12	LOAEL	Eisler (1988)

6.3.1.1 Arsenic

Arsenic is toxic to plants and can inhibit mitosis, photosynthesis, and respiration, and interfere with nucleic acid and protein synthesis. Arsenic toxicity is partially related to its bioavailability. Arsenic availability to plants from soil is highest in coarse-textured soils having little colloidal material and little ion exchange capacity. Except for highly polluted locations, arsenic generally is distributed throughout the plant in nontoxic amounts (Eisler, 1988a). At high concentrations, however, inorganic arsenic induces toxic effects primarily through a disruption of light activation and interference with metabolic pathways. Soil total arsenic concentrations of 25 to 80 mg/kg are sufficient to reduce crop yield in most plants (Eisler, 1988a), and arsenic soil concentrations as low as 15 mg/kg have been reported to be phytotoxic in some species (Kabata-Pendias and Pendias, 1984). This LOAEL is used to assess potential impacts on plants at NAS Alameda.

6.3.1.2 Cadmium

Cadmium in soil is absorbed passively by plants and translocated freely within the plant. Its phytotoxicity is related to alteration of cell membrane permeability and at least some toxic effects are linked specifically to interference of zinc-dependent uptake and translocation processes (Foy et al., 1978). Chlorosis is one of the general symptoms of cadmium toxicity in plants and appears to be caused by direct or indirect interaction of cadmium with foliar iron (Foy et al., 1978). A soil cadmium concentration of 3 mg/kg has been reported to be phytotoxically excessive in some plant species (Kabata-Pendias and Pendias, 1984). This LOAEL will be used to assess potential impacts to plants from cadmium in soil.

6.3.1.3 Chromium

The chromium content of plants is controlled mainly by the amount of soluble chromium in the soils. Chromium (VI) is the most soluble and available to plants, but it is also the most unstable form under normal soil

conditions. Usually chromium distribution in plants results in the highest concentrations in the roots, then the leaves and stems, and the lowest concentrations in the grain (Kabata-Pendias and Pendias, 1984). Typical symptoms of chromium phytotoxicity are wilting of plant tops, root injury, chlorosis in young leaves, brownish-red leaves, and chlorotic bands on cereals (Kabata-Pendias and Pendias, 1984). Concentrations as low as 0.01 mg/kg in the soil were found to cause a reduction in the dry weight of bean leaves (NAS, 1974). This LOAEL of 0.01 mg/kg is therefore used in this analysis to assess adverse impacts to plants.

6.3.1.4 Copper

Furr et al. (1978) and Dowdy and Larson (1975) conducted studies on plant growth in soils amended with sludge and reported soil/sludge copper concentrations of 395 mg/kg and 245 mg/kg had no effect on plant growth or physiology. However, Kabata-Pendias and Pendias (1984) reported soil copper levels as low as 60 mg/kg were phytotoxic to some plants. This value is selected for this assessment.

6.3.1.5 Lead

Lead inhibits plant growth, reduces photosynthesis, and reduces mitosis and water absorption (Eisler, 1988b). Inhibition of photosynthesis is attributed to the blocking of protein sulfhydryl groups and to changes in phosphate levels in cells (Eisler, 1988b). Lead levels of approximately 500 mg/kg soil reduced pollen germination by greater than 90% in two species of weeds (Eisler, 1988b). Normal germination rates were observed at soil lead levels of 46 mg/kg but other adverse effects were observed at lead levels of 12 to 312 mg/kg soil (Eisler, 1988b). The 12 mg/kg LOAEL is selected for this assessment.

6.3.2 Toxicity to Terrestrial Wildlife

Toxicity criteria for the protection of terrestrial wildlife have not been developed by regulatory agencies and therefore are developed in this section for the previously selected receptors and chemicals. The toxicity criteria derived for this preliminary assessment are derived using the highest doses associated with no adverse effect (NOAELs). If NOAELs are not available in the literature, the lowest LOAEL was used. When available, toxicity criteria are derived from studies with wildlife species or species similar to the selected indicator species. Phylogenetic similarities are used as the criterion for selection from higher taxonomic levels. Data on chronic or subchronic toxicity were used whenever available.

Uncertainty factors were applied to each NOAEL or LOAEL to derive the toxicity criteria. For example, a safety factor of 10 is applied to a LOAEL derived from a chronic study, and a safety factor of 100 is applied to a LOAEL derived from a subchronic study. An additional safety factor of 10 is applied to all toxicity values determined for species different than the receptor species at the site. Identical safety factors are applied in the derivation of human health toxicity criteria, and these factors are deemed similarly appropriate for the protection of wildlife species.

For those compounds for which only acute lethality values (e.g., LD_{50}) for wildlife are available, toxicity values for this assessment are derived by dividing the acute toxicity value by an uncertainty factor. In evaluating the potential effects of pesticides on wildlife, EPA analyzed a subset of available dose-response data and suggested that if the estimated dose is less than one-fifth of the median lethal dose for nonendangered species, no acute hazard can be presumed (Urban and Cook, 1986). We adopt this rule for this risk assessment, and derive acute toxicity values for terrestrial receptors by dividing acute lethality values by 5. As was the case for chronic data, an additional safety factor of 10 is applied to toxicity values derived from species different than the indicator species. This approach does not consider chronic toxicity.

Mammalian toxicity data are presented for arsenic, cadmium, chromium, lead, mercury, nickel, and PAHs. Avian toxicity data are presented for cadmium, chromium, copper, lead and zinc. Toxicity is discussed below by receptor. The toxicity values derived for mammals and birds are summarized in Tables 6-3 and 6-4, respectively.

6.3.2.1 Toxicity to Mammals

Arsenic. Arsenic poisoning in most animals is usually manifested by acute or subacute signs; chronic poisoning is rare because detoxification and excretion of small amounts of arsenic (below acutely or subacutely toxic doses) is rapid. General signs of arsenic toxicosis include peripheral nervous system disturbance, cardiac abnormalities, gastrointestinal disturbance, and death.

Quantitative data on the toxicity of arsenic to terrestrial wildlife species are limited, but arsenic has been shown to induce death in wild rabbits and hares following acute oral exposures. Median lethal doses (LD_{50}s) have been reported in the range of 10.5 to 40.4 mg/kg body weight (NRCC, 1977). A toxicity criterion of 2 mg/kg is derived by applying a safety factor of 5 to the lowest reported LD_{50} for rabbits.

Cadmium. Toxic effects of cadmium in laboratory animals include decreased growth rates, anemia, infertility, fetal abnormalities, abortions, kidney disease, intestinal disease, and hypertension. Acute oral LD_{50}s of 250 and 150 mg/kg were reported in rats and guinea pigs, respectively (Eisler, 1985). A single oral dose of 43 mg/kg of cadmium was lethal to rabbits (NAS, 1980). Applying an uncertainty factor of 5 (for estimating safe acute doses) to the lowest LD_{50}, an acute toxicity criterion of 8.6 mg/kg is derived.

Chromium. Acute and chronic toxicity of chromium is caused primarily by chromium (VI); elemental chromium and chromium (III) compounds are relatively nontoxic. Chromium toxicity is manifested by altered enzyme activity and blood chemistry, behavioral modifications, and histopathology. Studies on

TABLE 6-3

CRITICAL TOXICITY VALUES FOR THE ASSESSMENT OF IMPACTS TO HERBIVOROUS MAMMALS AT NAS ALAMEDA

Chemical	Critical Toxicity Value (mg/kg bw) (a)	Basis for Value					
		Species	Exposure Duration	Dose (b)	Effect	Source	Uncertainty Factor (c)
Arsenic	2	Rabbits and hares	Single dose	10.5	Death	NRCC 1977	5
Cadmium	8.6	Rabbits	Single dose	43	Death	NAS 1980	5
Chromium	0.5	Rats	Subchronic	1,000 ppm (diet)	NOAEL	Eisler 1986	100
Lead	0.0005	Rabbits	Subacute	0.005	Reduced blood ALAD activity	Eisler 1988	10
Mercury	0.013	Rats	2 years	2.5 ppm (diet)	NOAEL	Fitzhugh et al. 1950	10
Nickel	0.5	Rats	2 years	100 ppm (diet)	NOAEL	NAS 1980	10
PAHs	0.8	Deer Mice	5 days	825	Decreased food intake	Eisler 1987	1,000

(a) See text for derivation of values.

(b) Dose in mg/kg bw, unless otherwise noted.

(c) See text for description of uncertainty factors used in this assessment.

TABLE 6-4

CRITICAL TOXICITY VALUES FOR THE ASSESSMENT OF IMPACTS TO LAND-FORAGING BIRDS AT NAS ALAMEDA

Chemical	Critical Toxicity Value (mg/kg bw) (a)	Basis for Value					
		Species	Exposure Duration	Dose	Effect	Source	Uncertainty Factor (b)
Cadmium	0.0005	Black Ducks	4 Months	4 ppm (diet)	Altered behavior	Heinz and Haseltine (1983)	1000
Chromium	0.004	Black Ducks	5 Months	10 ppm (diet)	Altered growth and decreased survival	Heinz and Hazeltine (1981)	100
Copper	0.04	Turkeys	21 Days	50 ppm (diet)	NOAEL	NAS (1980)	100
Lead	0.12	American Kestrels	7 Months	50 ppm (diet)	NOAEL	Pattee (1984)	10
Zinc	0.13	Japanese Quail	2 Weeks	62.5 ppm (diet)	NOAEL	NAS (1980)	100

(a) See text for derivation of values.

(b) See text for description of uncertainty factors used in this assessment.

chromium toxicity in mammalian wildlife species were not identified in the literature. A dietary concentration of 1,000 ppm was the toxic threshold in rats (Eisler, 1986). Applying a dietary conversion factor of 0.05 kg food/kg body weight for rats (USDA, 1984), the threshold dietary level corresponds to a dosage of 50 mg/kg body weight. Applying a safety factor of 100 (10 for interspecies differences and 10 for subchronic exposures) to this value gives a toxicity criteria of 0.5 mg/kg for rabbits.

Lead. Lead adversely affects survival, growth, development, and metabolism of most animal species. Acute toxicosis is often characterized by impairment of the central nervous system, the gastrointestinal tract and the muscular and hematopoietic systems and death. Most data on the toxic effects of lead in mammals are from studies with laboratory and domestic species. In wildlife species, subacute exposure of rabbits to lead dosages of >0.005 mg/kg reduced blood ALAD activity and dosages of 0.03 mg/kg resulted in elevated blood levels (Eisler, 1988). A toxicity criterion of 0.0005 mg/kg is derived by applying an uncertainty factor of 10 (for using a subchronic study) to the LOAEL of 0.005 mg/kg. An additional uncertainty factor of 10 for extrapolation of a NOAEL from a LOAEL is not used here because the effect (altered blood ALAD) is regarded as minimally severe.

Mercury. Mercury poisoning in mammals is manifested by a variety of effects. At comparatively low concentrations in mammals, it adversely affects reproduction, growth and development, behavior, blood and serum chemistry, motor coordination, vision, histology, and metabolism (Eisler, 1987). Most data on the mammalian toxicity of mercury are limited to methylmercury studies in laboratory species. In laboratory studies with inorganic mercury, no adverse effects were observed in rats exposed to 2.5 ppm mercury in the diet for 2 years (Fitzhugh et al., 1950). Applying a dietary conversion factor of 0.05 kg food/kg body weight for rats (USDA, 1984), this dietary NOAEL corresponds to a dosage of 0.13 mg/kg. A toxicity criterion of 0.013 mg/kg day is derived for rabbits using the NOAEL and an uncertainty factor of 10 (for interspecies differences).

Nickel. In laboratory animals, nickel causes a variety of toxic effects, including toxic effects in the kidney, spleen, bone, and blood. Rats exposed for 2 years to dietary nickel at a concentration of 100 ppm showed no adverse effects, although higher dietary concentrations resulted in decreased growth, weight loss, and liver and blood effects (NAS, 1980). Using a dietary conversion factor of 0.05 kg food/kg body weight for rats (USDA, 1984), this diet concentration corresponds to a daily dosage of 5 mg/kg. A toxicity criterion of 0.5 mg/kg is derived by applying a safety factor of 10 (for interspecies differences) to the NOAEL.

PAHs. The primary effect of PAH exposure in laboratory test animals is tumor development. In wild species, food consumption in deer mice decreased following oral exposure for 5 days to 825 mg/kg bw of 2-methoxynaphthalene, a noncarcinogenic PAH (Eisler, 1987). Applying a safety factor of 1000 (100 for use of a LOAEL from a less than chronic study and 10 for interspecies differences) to this value gives a toxicity criterion of 0.8 mg/kg.

6.3.2.2 Toxicity to Birds

Cadmium. Cadmium can cause death and a variety of sublethal effects in birds exposed via the diet. Sublethal effects include growth retardation, anemia, and testicular damage. In mallard ducklings fed dietary cadmium from day 1 of age for 12 weeks, hematological effects were observed at 8 weeks and mild to severe kidney lesions developed at 12 weeks (Cain et al., 1983). However, no hematological effects were observed in adult mallards exposed to 20 or 200 ppm cadmium in the diet for 90 days (White and Finley, 1978), although kidney lesions, testicular degeneration, and complete cessation of spermatogenic activity were observed in adult mallards exposed to 200 ppm in the diet for 90 days (White et al., 1978). In other duck species, no effects on growth or kidneys were observed in wood ducks exposed from age 1 week to age 7 weeks to 1, 10, or 100 ppm cadmium in the diet (Mayack et al., 1981). Altered avoidance behavior in the form of hyperresponsiveness was observed in young black ducks born to parents exposed to 4 ppm cadmium in the diet for 4 months prior to birth of their young (Heinz and Haseltine, 1983). Assuming

(by analogy to chickens; Welty, 1982) a duck consumes an amount of food daily that is equal to 4% of its body weight, the dietary LOAEL corresponds to a dosage of 0.16 mg/kg day. A toxicity value of 0.0002 mg/kg is derived by applying an uncertainty factor of 1000 (for using a LOAEL from a subchronic study with a different species) to the LOAEL.

Chromium. Adult black ducks fed diets containing 10 or 50 ppm chromium (III) for five months were normal in survival, reproduction, and blood chemistry (Haseltine et al., 1985). However, ducklings from treated groups that were fed chromium at the original parental doses experienced altered growth and reduced survival. No effect on avoidance response was observed in young black ducklings fed chromium at 20 or 100 ppm in the diet for seven days that were born to parents fed these same doses (Heinz and Haseltine, 1981). The 10 ppm dietary LOAEL in ducks was the lowest LOAEL for birds indicated in the available literature. Because no toxicity data on chromium (VI) were available, the toxicity of chromium (III) is used as representative of chromium (VI) toxicity. Assuming a duck ingests an amount of food daily equal to 4% of its body weight (by analogy to domestic chickens; Welty, 1982), the dietary LOAEL corresponds to a dosage of 0.4 mg/kg day. Applying an uncertainty factor of 100 (for using a LOAEL and for interspecies differences) a toxicity criterion of 0.004 mg/kg is derived.

Copper. Copper caused reduced growth in young turkeys exposed to 100 ppm copper in the diet for 21 days, although dietary concentrations of 50 ppm had no adverse effects (NAS, 1980). Copper at a concentration of 100 ppm in the diet of ducks resulted in increased growth (NAS, 1980). The 50 ppm dietary NOAEL from the studies with turkeys will be used to form the basis of the toxicity criterion for this study. Assuming a turkey ingests an amount of food daily equal to 4% of its body weight (by analogy to domestic chickens, Welty, 1982), the dietary NOAEL corresponds to a dosage of 4 mg/kg day. Applying an uncertainty factor of 100 (for using data from less than chronic exposures and for interspecies differences), a toxicity criterion of 0.04 mg/kg is derived.

Lead. The majority of information on lead toxicity in birds is on body burdens in waterfowl that have ingested spent lead shot and died. Dose-response information is additionally available for a few species. Neurological effects were observed within 24 hours of dosing in mallard ducks that had ingested and absorbed lead shot for a total intake of 423.8 mg/kg body weight (Mautino and Bell, 1987). In 1-day-old American kestrels fed 125 or 625 mg/kg body weight lead for 10 days, growth was seriously depressed by day 6, and hematocrit values were significantly depressed by day 10 (Hoffman et al. 1985). Forty percent of the birds receiving 625 mg/kg lead died within 6 days. No effects were observed in kestrels exposed to 25 mg/kg body weight. American kestrels fed 10 or 50 ppm lead in the diet for 7 months experienced no toxic effects with respect to survival, egg laying, initiation of incubation, or egg shell thickness (Pattee, 1984). Assuming a kestrel weighs 0.11 kg and ingests 26 g of food each day (USDA, 1988), these dietary levels correspond to dosages of 2.4 and 12 mg/kg body weight. The 12 mg/kg NOAEL is the highest chronic NOAEL for birds based on the studies reviewed and is used to develop a toxicity criterion for this assessment. An uncertainty factor of 10 (for interspecies differences) is applied to the NOAEL to derive a criterion of 0.12 mg/kg.

Zinc. In experimental animals, zinc has been reported to cause bone and blood abnormalities, reduced weight gain, anemia, and decreased tissue concentrations of some minerals. Ducks fed zinc in the diet for 60 days at concentrations of 3,000 to 12,000 ppm experienced decreased food consumption and loss of body weight, paralysis of legs, altered organ weights, and low hemoglobin and hematocrit (NAS, 1980). Japanese quail exposed to 125 ppm to 2,000 ppm dietary zinc for 2 weeks experienced decreased hemoglobin and hematocrit as well as other toxic effects (NAS, 1980). No toxic effects were observed at a dietary concentration of 62.5 ppm. Assuming a quail weighs 170 g and eats 34 g of food each day (USDA, 1988), the 62.5 ppm dietary level corresponds to a dosage of 12.5 mg/kg body weight. A toxicity criterion of 0.13 mg/kg is derived by applying an uncertainty factor of 100 (for less than chronic exposures and for interspecies differences).

6.3.3 Toxicity to Aquatic Life

Toxicity criteria for the protection of aquatic life have been developed by EPA and the State of California for some of the chemicals in the groundwater and sediment at NAS Alameda. These ambient water quality criteria are used in this preliminary assessment and are presented in Table 6-5.

6.4 ASSESSMENT OF RISKS

In this section, potential risks to plants, terrestrial wildlife, and aquatic life at NAS Alameda are discussed. Discussions are primarily qualitative because the available monitoring data are not adequate to assess exposure to most receptors. Monitoring data needed to assess risks also are presented.

6.4.1 Plants

Data from the shallow soils (0 to 1 feet) of the 1943-1956 Disposal Area and the West Beach Landfill are needed to characterize potential chemical-induced impacts to plants at NAS Alameda. Potential impacts could be estimated by comparing the concentrations of the chemicals in the shallow soils (within the root zone) with the concentrations known to be phytotoxic in at least some species (presented in Table 6-2). If soil levels exceed phytotoxic levels, adverse effects may occur.

The nature of the toxic effects which may occur is difficult to predict. Because the majority of the landfill and disposal areas are vegetated, it is apparent that the chemicals in the soil are not at lethal concentrations, at least for some species. More likely, if chemical-induced effects are occurring, the soil contaminants may be causing reduced growth or reproduction or otherwise altering the physiology of the plants that are present at the site. Toxic effects in some species may result in changes in the composition and structure of the plant community as species not as sensitive to the soil

TABLE 6-5

WATER QUALITY CRITERIA FOR THE PROTECTION OF AQUATIC LIFE (a)

Chemical	California AAL (b)	Federal AWQC (c)	
		Acute	Chronic
Aldrin	NC	1.3	NC
Arsenic	22.4	69	36
Cadmium	5.1	43	9.3
Chromium	1.53	1,100	50
Copper	6	2.9	2.9
DDT	NC	0.13	0.001
Heptachlor	NC	0.053	0.0036
Lead	4.4	140	5.6
Lindane	NC	0.16	NC
Mercury	NC	2.1	0.025
Nickel	NC	75	8.3
PCBs	NC	10	0.03
Selenium	NC	410	54
Silver	NC	2.3	NC
Zinc	12	95	86

(a) Criteria are for marine species.

(b) Applied action level for aquatic species.

(c) Federal Ambient Water Quality Criterion. Acute

NC = Criterion not developed.

contaminants may become dominant. This could result in changes in the habitat type and quality of the area.

6.4.2 Terrestrial Wildlife

6.4.2.1 Jackrabbits and Other Herbivorous Mammals

Data from the shallow soils of the West Beach Landfill are needed characterize potential chemical-induced effects associated with ingestion of plants by jackrabbits or other herbivorous mammals at NAS Alameda. Data from shallow soils are necessary because contamination in these soils is representative of the contamination to which plants would be exposed. Potential impacts could be estimated by comparing critical toxicity values with the estimated dietary intake of chemicals by jackrabbits or other mammals ingesting vegetation that has accumulated soil contaminants. Estimated chemical intakes that exceed critical toxicity values may be associated with adverse impacts in jackrabbits and other herbivorous mammals at NAS Alameda.

Based on the potential contaminants at NAS Alameda, exposure could affect survival, growth, development, and metabolism of jackrabbits and other herbivorous mammals at NAS Alameda. Continual impacts on survival obviously could reduce the jackrabbit (herbivore) population at the NAS Alameda. This, in turn, could affect other species that rely on rabbits or other herbivores for food. In addition, these animals could serve as the route by which these predators become exposed to soil contaminants.

6.4.2.2 Killdeer and Other Land-foraging Birds

Data from the shallow soils of the West Beach Landfill and the 1943-1956 Disposal Area are needed characterize potential chemical-induced effects in killdeer (or other land-foraging birds). Estimates of impact could be derived by comparing estimated dietary intake to the critical toxicity values derived for birds. Based on the potential contaminants at NAS Alameda, exposure could possibly result in a variety of toxic effects, including decreased growth

(copper, lead), decreased survival (chromium, lead), altered behavior (cadmium), and altered blood chemistry (zinc). Such effects could directly affect the health of the killdeer or other land-foraging bird populations at NAS Alameda. Continual reductions in the survival of any species could result ultimately in the elimination of that species from the impacted area. It is possible, however, that immigration of animals from areas adjacent to the impacted area as habitat is vacated would be sufficient to replace lost individuals and maintain the population at NAS Alameda. Such a system is not ecologically stable however, as it requires continual inputs to maintain or approach equilibrium.

6.4.2.3 Plovers and Other Wetland and Shore Birds

The potential impacts to plovers and other wetland and shore birds cannot be evaluated at this time because information on contamination (type and quantity) in the wetland areas is not known. It is believed that the primary contaminants in the landfill area are lead, nickel, mercury, lindane, oil, naphthalene, toluene, acetone, and PCBs (Alliance/WESTDIV, 1987). The metals, lindane, and PCBs are known to bioconcentrate to some degree in aquatic invertebrates, which are the main food item of plovers and other wetland and shore birds. In addition, all the chemicals potentially present in the wetland and shore areas are potentially toxic to birds to some degree and can cause such effects as altered reproduction, neurological effects and death. The bioconcentration of chemicals in food items could lead to increased exposure and potentially increased impacts in the wetland and shore bird populations at NAS Alameda. Sediment and soil data from the wetland areas are needed before potential impacts can be assessed. In addition, sediment bioaccumulation studies could provide information on potential concentrations of contaminants in the food of plovers and other wetland and shore birds.

6.4.2.4 California Least Terns and Brown Pelicans (Bay-foraging Birds)

The potential impacts on the breeding population of California least terns and the fall and winter population of brown pelicans cannot be estimated because information on the concentration of chemicals in the waters in which these birds feed is not known. Potential contaminants in the Seaplane Lagoon include metals (mercury, lead, chromium, cadmium, zinc, nickel, copper, iron), pesticides (chlordane, DDT, 2,4-D, diazinon, lindane, malathion, and warfarin), oil, and PCBs. Again, some of these contaminants are known to bioconcentrate in fish (the food of terns and pelicans) and thus feeding in the waters surrounding NAS Alameda represents a pathway by which bay-foraging birds could be exposed.

The chemicals potentially occurring in the waters where the California least terns and brown pelicans feed are known to be toxic to bird species. Some of these chemicals (e.g., DDT, PCBs) can be associated with significant reproductive impact. Because both terns and pelicans are endangered species, any effects on the populations at NAS Alameda could impair the recovery of each species. This is particularly true for the California least tern, which breeds at NAS Alameda. The breeding colony at NAS Alameda is the largest colony in northern California. Toxic effects in this population thus could influence the survival of the species in northern California.

Recent studies on the tern colony indicate good reproductive success (measured as number of fledglings per nesting pair) of the breeding population (Collins, 1987). Other observations on adult and chick mortality suggest that the population is relatively healthy (Collins, 1987) and thus does not seem to be adversely affected by chemical contamination at NAS Alameda. However, the available information does not permit a complete evaluation of the health of the population because information is not available on, among other things, chick growth or on chick survival after leaving the nesting area. It is possible that contaminant-induced effects may become important only after the fledgling chicks must forage for themselves. (Past studies at NAS Alameda occurred at a time when chicks were still being fed by parents.) For

example, studies on the effects of lead in common tern chicks indicate that lead can impair the ability of the chick to manipulate fish during feeding, a behavior which involves a complex motor coordination of the head and neck (Gochfeld and Burger, 1988). Such effects could become significant when chicks leave the nesting area and must forage for themselves. Reduced feeding efficiency and food intake could result in decreased body weight gain. Even such subtle impairment of feeding behavior and/or slowing of normal growth could have significant effects on survival under natural conditions in which energy requirements and stress could exacerbate toxic effects. Such effects could have a particulary significant impact on young California least terns which must migrate from NAS Alameda to wintering grounds, an activity which requires high energy reserves. Birds unable to forage effectively may suffer reduced survival.

More data are needed on the concentration of contaminants in fish or in the water surrounding NAS Alameda where the terns and pelicans feed before a more detailed assessment of potential impacts on these species can be performed.

6.4.3 Aquatic Life

Potential effects in aquatic species were evaluated conservatively by comparing measured chemical concentrations in groundwater and estimated chemical concentrations in sediment pore water with aquatic criteria for the protection of aquatic life (Table 6-5). Tables 6-6 and 6-7 present these comparisons for groundwater and sediment pore water, respectively. As Table 6-6 indicates, the measured concentrations of chemicals in the groundwater at NAS Alameda exceed the California AAL and/or the Federal AWQC for the protection of aquatic life. It is possible, therefore, that aquatic organisms may be adversely impacted by groundwater contaminants at NAS Alameda. Exposures, and therefore risks, would be greatest for sessile benthic species living at the discharge point; mobile species (e.g., fish) and benthic species living further from the discharge point would be exposed to more dilute concentrations. Continual impairment of aquatic species at NAS Alameda could

TABLE 6-6

COMPARISON OF GROUNDWATER CHEMICAL CONCENTRATIONS WITH CALIFORNIA AALs AND FEDERAL AMBIENT WATER QUALITY CRITERIA FOR THE PROTECTION OF AQUATIC LIFE

(Concentrations in ug/liter)

Chemical	AAL	AWQC		Groundwater Concentration (a)			
		Acute	Chronic	Landfill	1943-1956 Disposal Area	Area 97	CANS Area
Aldrin	NC	1.3	NC	0.1 (b)	---	---	---
Arsenic	22.4	69	36	43	---	---	---
Cadmium	5.1	43	9.3	32	---	---	---
Chromium	1.53	1,100	50	104	---	---	130
Copper	6.0	2.9	2.9	63	---	---	---
DDT	NC	0.13	0.001	0.7 (b)	---	---	---
Heptachlor	NC	0.053	0.0036	0.3	---	---	---
Lead	4.4	140	5.6	186	---	1,200	---
Lindane	NC	0.16	NC	0.3 (b)	---	---	---
Mercury	NC	2.1	0.025	1.3	---	---	---
Nickel	NC	75	8.3	120	---	---	---
PCBs	NC	10	0.03	8	---	---	---
Selenium	NC	410	54	42	---	---	---
Zinc	NC	95	86	64	130	---	---

(a) Geometric mean value, unless otherwise noted.
(b) Maximum value.

--- = Chemical not detected or not analyzed for.

TABLE 6-7

COMPARISON OF ESTIMATED SEDIMENT PORE WATER CHEMICAL CONCENTRATIONS WITH FEDERAL AWQCs AND CALIFORNIA AALs FOR PROTECTION OF AQUATIC LIFE

(Concentrations in ug/liter)

Chemical	AAL	AWQC		Estimated Sediment Pore Water Concentration	
		Acute	Chronic	Sea Plane Lagoon	Oakland Inner Harbor
Arsenic	22.4	69	36	1,100	1.200
Cadmium	5.1	43	9.3	230	65
Chromium	1.53	1,100	50	2,500	3,300
Copper	6.0	2.9	2.9	940	1,300
Lead	4.4	140	5.6	34	24
Mercury	NC	2.1	0.025	18	39
Nickel	NC	75	8.3	850	1,500
Selenium	NC	410	54	1,800	ND
Silver	NC	2.3	NC	ND	2.2
Zinc	12	95	86	1,800	2,700

NC = No criterion developed.

ND = Chemical not detected in sediments of this area.

result in decreased populations of many fish and invertebrate species. The Bay waters are important areas for juvenile fish and toxic effects in juveniles as well as adults could result in decreases in the sport and commercial fisheries of the area.

The analysis used here to evaluate potential impacts on aquatic life at NAS Alameda is extremely conservative and therefore should be viewed strictly as a screening method. There is much uncertainty currently surrounding the movement of groundwater at NAS Alameda and the dilution of contaminants which may enter the Bay waters, as well as uncertainty surrounding the available monitoring data which preclude more accurate predictions at this time.

Comparison of estimated sediment pore water with aquatic toxicity criteria also reveals the potential for adverse effects in the Seaplane Lagoon and the Oakland Inner Harbor as the estimated concentrations of all detected chemicals exceed at least one criterion. The approach used here to estimate exposure should however be regarded as a screening approach and as an indicator of potential problems because of the uncertainties intrinsic to using Kd values to determine partitioning of inorganic chemicals to water in the sediments. Because Kds are affected by a variety of factors, including metal speciation and sediment organic carbon and hydrous metal oxide content, generic partitioning approaches are as accurate for inorganics as for organics. Sediment bioassays are the best method of determining sediment toxicity at NAS Alameda and should be performed on sediment from the Seaplane Lagoon and Oakland Inner Harbor, as well as on sediments from the on-site wetlands and control sites.

The above sediment analysis does not evaluate potential benthic impacts associated with exposure to sediment-bound tributyltin and other butyltins. Although sediments from the Seaplane Lagoon were not analyzed for butyltins, they may contain these compounds since antifouling paints containing tributyltin were used on boats berthed in the Seaplane Lagoon. Laboratory and field tests have demonstrated that tributyltin and its degradation products

(di and monobutyltin) are toxic to nontarget aquatic organisms at low environmental concentrations, potentially affecting growth, development, and reproduction in exposed organisms. Sensitivity to tributyltin varies among aquatic species with gastropods and bivalves being the most susceptible followed by crustaceans, algae, and fish (Rexrode, 1987). Recent studies on butyltin in marine systems suggest that sediments act as a sink for tributyltin and may be a long-term chronic source of tributyltin and its degradation products even after tributyltin inputs cease (Wade et al., 1988). Given this potential persistence and the known toxicity of these compounds, the tributyltin and butyltin content of sediments from the Seaplane Lagoon should be determined during the RI sampling.

6.4.4 Conclusions

The preliminary environmental assessment of NAS Alameda indicates a potential for adverse effects in plants, mammals, birds, and aquatic life. More complete estimates of potential impacts cannot be made at this time because of the lack of adequate sampling data to estimate contamination and potential exposure.

Additional data collected during the remedial investigation will permit more complete evaluations of potential impacts. Monitoring data from the shallow soils (0 to 1 foot) of the 1943-1956 Disposal Area and the West Beach Landfill are needed to assess potential impacts on plants, rabbits (and other herbivorous mammals), and killdeer (and other land-foraging birds) in these areas. Surface water and sediment samples are needed from the on-site wetlands and the surrounding Bay waters, including the Seaplane Lagoon and the Oakland Inner Harbor. Sediment and surface water bioassays and analysis of fish tissues from the Bay waters and sediment bioaccumulation studies for the on-site wetlands would also facilitate the final environmental assessment.

The sampling activities conducted as part of the remedial investigation are expected to have no significant impact on the ecosystem of the NAS Alameda area. Sediment sampling will result in some disturbance of the benthic

community in the sampled area (e.g., displacement of sediment and organisms), however, the area impacted will be small (less than a square meter) relative to the size of unsampled areas so that the overall impact on the benthic community of the area will be negligible. Similarly, negligible impacts are expected on the terrestrial soil community as a result of soil sampling and the construction of groundwater wells. Collection of fish and benthic organisms for tissue residue analyses will result in the removal of individuals from the aquatic populations of the area. Again, however, because the number of organisms removed from the study area will be small relative to the total number of organisms present in the waters on and surrounding NAS Alameda, the overall impact on the aquatic community of the area will be negligible. No impacts are expected to be associated with the surface water or groundwater sampling.

7.0 IDENTIFICATION OF DATA GAPS

Preliminary studies conducted at the NAS Alameda site have identified a number of areas where hazardous chemicals have been used or released. Sampling data are available for only 7 of the 20 areas identified by Alliance/WESTDIV (1987). In order to quantitatively evaluate the potential risks to human health and the environment from these sites, additional information is required on the identity and distribution of chemicals at the site, the routes by which chemicals could potentially be transported to and within surface waters, and the potential human and environmental receptor populations. The RI Sampling and SWAT plans prepared by Canonie/WESTDIV (1988a; 1988b) address many of the data needs for a comprehensive public health assessment. The following discussion first outlines the general data needs for each medium (e.g., groundwater). Specific data requirements for individual areas at the site are shown in Table 7-1.

7.1 CHEMICAL CHARACTERIZATION

Preliminary investigations conducted at the site have identified a number of areas where contaminants are present in the soil, the underlying groundwater, or in the form of vapors or gases. Other areas are thought to have been affected by chemical releases based on site history or visible contamination. The nature of the chemical contamination has not been adequately characterized in each study area. The areal extent affected by each source of contamination and the direction and extent of migration from the source have not been thoroughly evaluated.

The initial chemical characterization of each source area should include analyses for non-priority as well as priority organics, since many common environmental contaminants (e.g., acetone) are not on the EPA priority pollutant list. Additional chemicals which should be tested for, but are not on the priority pollutant list include certain pesticides (diazinon, malathion), herbicides (bromacil, monuron, diuron, glyphosate, simazine), radionuclides, and explosives and their byproducts (not identified in

Table 7-1 Summary of Chemical Characterization Data Required for the Public Health and Environmental Evaluations

Site Area	Surface Soil[a]	Soil Borings	Groundwater	Sediment	Surface Water	Air
Maintenance area (Bldg. 41)	BNA, M[b]	VOA, BNA, M, P	VOA, BNA, M, P			
Oil and Gasoline areas						
Bldgs. 459, 547, 162 (Service Stations)	BNA, M, P	VOA, BNA, M, P	VOA, BNA, M, P			VOA
Bldg. 10 (Power Plant)	BNA, M, P	VOA, BNA, M, P	VOA, BNA, M, P			VOA
Area 97	BNA, M	VOA, BNA, M	VOA, BNA, M			VOA
Oil Refinery	BNA, M, P	VOA, BNA, M, P	VOA, BNA, M, P			VOA, BNA, M
Fire Training Area	BNA, M, P, D	VOA, BNA, M, P	VOA, BNA, M, P			VOA, BNA, M, P
Building 114	BNA, M, P	VOA, BNA, M, P	VOA, BNA, M, P			

a - surface soils from 0-2" below ground surface
b - underlined parameters were not included in Canonie sampling plan
VOA - volatile organic analysis
BNA - base/neutral/acids extractables (priority and non-priority organics, including PAHs)
M - metals (includes priority and non-priority metals)
P - pesticides/PCBs and herbicides (PCBs as Aroclor mixtures)
CN - total cyanide
D - polychlorinated dibenzodioxins/dibenzofurans (total each homolog group plus 2,3,7,8-tetra dioxin and furan)
I - Indicator parameters (pH, TOC, Cl)
R - Radioactivity (gross alpha and beta)
H - Hardness
O - Oil and grease
A - Asbestos

Table 7-1 Summary of Chemical Characterization Data Required for the Public Health and Environmental Evaluations (continued)

Site Area	Surface Soil[a]	Soil Borings	Groundwater	Sediment	Surface Water	Air
Paint stripping and plating areas						
Bldg. 5	BNA, M, CN, A	VOA, BNA, M, CN, A	VOA, BNA, M, CN			VOA, M
Bldg. 360	BNA, M, CN	VOA, BNA, M, CN	VOA, BNA, M, CN			VOA, M
Bldg. 410	BNA, M	VOA, BNA, M	VOA, BNA, M			VOA, M
Bldg. 400, 530 (Missile rework areas)	BNA, M	VOA, BNA, M	VOA, BNA, M			VOA, M
Test shop (Bldg. 14)		VOA, BNA, M	VOA, BNA, M			
Transformer Storage areas (Bldgs. 301, 389)	BNA, P, M	VOA, BNA, M, P	VOA, BNA, M, P			M, P
Waste Storage area (CAN C-2 Area)	BNA, M, P, CN	VOA, BNA, M, P, CN	VOA, BNA, M, P, CN			VOA, BNA, M, P
Marine Environments						
Seaplane Lagoon				VOA, BNA, M, P	VOA, BNA, M, O, P	
Oakland Estuary				VOA, BNA, M, P	VOA, BNA, M, O, P	
1943-1956 Disposal Area	BNA, M, P, R, A	VOA, BNA, M, P, R, A	VOA, BNA, M, P, R, I, A			VOA, BNA, M

a - surface soils from 0-2" below ground surface
b - underlined parameters were not included in Canonie sampling plan
VOA - volatile organic analysis
BNA - base/neutral/acids extractables (priority and non-priority organics, including PAHs)
M - metals (includes priority and non-priority metals)
P - pesticides/PCBs and herbicides (PCBs as Aroclor mixtures)
CN - total cyanide
D - polychlorinated dibenzodioxins/dibenzofurans (total each homolog group plus 2,3,7,8-tetra dioxin and furan)
I - Indicator parameters (pH, TOC, Cl)
R - Radioactivity (gross alpha and beta)
H - Hardness
O - Oil and grease
A - Asbestos

Table 7-1 Summary of Chemical Characterization Data Required for the Public Health and Environmental Evaluations (continued)

Site Area	Surface Soil[a]	Soil Borings	Groundwater	Sediment	Surface Water	Air
West Beach Landfill	BNA, P, M, R, A	VOA, BNA, M, P, R, A	VOA, BNA, M, P, R, I			
Yard D-13	BNA, P, M, CN	VOA, BNA, M, P, CN	VOA, BNA, M, P, CN			VOA
Wetland areas				VOA, BNA, M, P	VOA, BNA, M, P, O, H	

a - surface soils from 0-2" below ground surface
b - underlined parameters were not included in Canonie sampling plan
VOA - volatile organic analysis
BNA - base/neutral/acids extractables (priority and non-priority organics, including PAHs)
M - metals (includes priority and non-priority metals)
P - pesticides/PCBs and herbicides (PCBs as Aroclor mixtures)
CN - total cyanide
D - polychlorinated dibenzodioxins/dibenzofurans (total each homolog group plus 2,3,7,8-tetra dioxin and furan)
I - Indicator parameters (pH, TOC, Cl)
R - Radioactivity (gross alpha and beta)
H - Hardness
O - Oil and grease
A - Asbestos

preliminary investigations). Dioxins/furans should be analyzed for in areas where PCBs may have been burned (e.g., Fire Training area). Each organic analysis should also include identification by GC/MS of the 10 largest unidentified chromatographic peaks. Subsequent rounds of sampling may then be limited to those compound classes that have been shown to be present in the initial samples.

7.1.1 Surface Soils

In general, surface soil samples should be obtained from all areas where soil contamination is known or suspected to exist. Surface soil samples should be obtained from the upper 2" of the soil column, to obtain an accurate estimate of those chemicals that are strongly absorbed to soil (e.g., metals, PCBs). Inclusion of deeper soils could dilute the concentration of these chemicals, and therefore lead to underestimation of exposure point concentrations. Surface soil information is also important for evaluation of potential impacts on the ecological communities in the wetlands on the site, for estimation of dust generation, and for estimation of storm runoff to the Seaplane Lagoon and Oakland Inner Harbor.

Background soil samples are generally necessary for evaluating potential risks from a site, and selection of appropriate background samples is important to the conclusions of a public health assessment. A minimum of three background samples are required for each stratum sampled. Background samples should not be composited and should be spatially distributed in order to characterize background concentrations. Background sample selection may be difficult for soils that are composed entirely of fill material.

If sampling confirms the existence of widespread or high levels of surface soil contamination, physical and chemical properties of the site soils should be determined. Soils should be analyzed for pH, organic carbon content (f_{oc} or TOC), to aid in evaluation of leaching potential.

In addition, a representative selection of surface soil samples should be subjected to sieve analysis for silt content, in order to estimate the potential for emission of respirable particulates from the site. Topographic factors affecting wind and water erosion should also be evaluated. These factors include soil bulk density and erodibility; surface slope, roughness; and percent of site soils covered by asphalt, buildings, or vegetation.

To estimate the potential risks from inhalation of chemicals by persons living or working at the site, the pathways of migration of organic vapors and landfill gases through the soil should be thoroughly characterized. Data needs for estimating vapor transport include soil porosity, areas of impermeable cover (which can channel vapors), and zones of increased permeability (e.g., pipelines, filled trenches). In a complex, built-up area such as the NAS Alameda site, it is preferable to use ambient air sampling to evaluate the airborne exposure pathway, rather than to attempt to estimate exposure point concentrations from soils data.

7.1.2 Subsurface Soils

Soils beneath the NAS Alameda site are composed of heterogeneous fill and Bay Mud mud dredge materials. Therefore, the transport of chemicals in the subsurface may vary widely from one location to the next. The subsurface investigation should include testing of soils for physical/chemical parameters such as pH and TOC, in addition to the chemical analyses shown in Table 7-1.

7.1.3 Groundwater

Due to the proximity of the site to the Bay, much of the groundwater beneath the site is likely to be saline. A thorough understanding of the groundwater chemistry and hydrogeology of the site is needed, however, in order to evaluate potential risks to persons contacting off-site groundwater, and to evaluate potential environmental and public health impacts from groundwater discharging to surrounding surface waters. The direction of flow and recharge and discharge areas for groundwater should be determined in order

to identify impacted areas. For example, if the wetland in the West Beach Landfill is a discharge area for groundwater, it is important in determining the impact of contamination on wildlife using the wetlands as a feeding area. The hydrogeologic investigation should be designed to provide information on the location and volume of discharges to surface water bodies, which include the San Francisco Bay, the Seaplane Lagoon, and Oakland Estuary, and the potential for reversal of flow if groundwater was pumped to the east of the site. The hydrogeologic investigation should determine if a lens of potable fresh water exists beneath NAS Alameda. The interconnection of aquifers beneath the base with aquifers located off-site should also be addressed.

In addition to analyses to detect site-related chemicals, the indicator parameters pH, TOC, and dissolved oxygen should be measured, to evaluate fate and transport in the subsurface. Groundwater should also be analyzed for common inorganic species, including anions (sulfate, chloride, etc.) and cations (metals, calcium, etc.). This information would be essential if it became necessary to distinguish between site-related contamination and natural groundwater composition. For example, seawater intrusion may be recognized by the balance of cations even in the presence of excess chloride from a site.

The NAS Alameda site is situated on former marsh and bay fill, and the depth to groundwater is generally only a few feet. The groundwater gradients are therefore very small, and localized or seasonal reversals in the general westward flow may occur. It is important for the purposes of the ecological assessment, in particular, to determine the tidal and seasonal influences on groundwater flow patterns. The network of monitoring well pressure transducers proposed by Canonie/WESTDIV (1988a,b) will provide a continuous record of water levels, which can be used to derive this information.

In addition to the monitoring wells installed at the site, at least three background samples are required to allow a statistical comparison of site and background concentrations. For those inorganic chemicals which are naturally found in groundwater (e.g., some metals), comparison with background concentrations will be a factor in selection of chemicals to be considered in

the final public health evaluation. Background wells should be screened in a stratigraphically equivalent formation to sample wells.

7.1.4 Surface Water and Sediment: Marine Environments

Sediment samples were obtained in previous investigations from the Seaplane Lagoon and the Oakland Inner and Outer Harbors. The Seaplane Lagoon is a feeding ground for California least terns, and a thorough characterization of the surface water quality is needed to evaluate the potential risks to this species. It is recommended that surface water samples be obtained at all grid points in the Lagoon (Canonie/WESTDIV, 1988a). Additional surface water and sediment samples should be obtained from the vicinity of the storm drain outfalls in the Seaplane Lagoon and Oakland Inner Harbor, from locations of suspected groundwater discharge, and from the San Francisco Bay south of the NAS Alameda site. The detection limits for sediment and water samples should be adequate to detect chemicals below the Ambient Water Quality Criteria or California Applied Action Levels. PCBs in sediments should be detectable below 0.1 ppm in sediments in order to estimate potential adverse affects on the aquatic ecosystem. The organic carbon content of all sediment samples should be determined. Tin or tributyltin should be included in the initial list of metals analyzed in the sediments from the Sea Plane Lagoon.

Surface waters in the lagoon and harbor should be analyzed for metals and volatile organics. Even though volatiles would quickly be lost from the water, inputs from discharging groundwater could potentially maintain levels of concern. Surface waters should also be analyzed for oil and grease.

7.1.5 Surface Water and Sediment: Wetlands

Two small wetland areas exist on the site: in the vicinity of the West Beach Landfill, and to the west of the Seaplane Lagoon. To determine the potential risks to wildlife populations that live or feed in these wetlands, surface waters and sediments should be analyzed for metals, volatile and semi-

volatile organics, pesticides/PCBs, and oil and grease. Surface waters should also be tested for salinity and hardness. The organic content of sediment samples should be determined. It is also recommended that bioassays and bioaccumulation studies be performed with sediments from the wetland areas if elevated levels of contaminants with high bioconcentration factors (e.g., organochlorine pesticides, PCBs, and some metals) are found in the sediments.

7.1.6 Biota

Bioassays and tissue residue measurements are needed to evaluate potential impacts on the ecological communities of the area. Bioassays are useful for determining if chemicals present at the site are at concentrations that are toxic to the species that inhabit the waters of the NAS Alameda area. Tissue residue analyses are useful for determining if chemicals related to the site are accumulating in the food chain.

Bioassays

Canonie/WESTDIV (1988a) has proposed macrobenthos bioassays for the sediments of the Seaplane Lagoon and the Oakland Estuary. An evaluation of sediment toxicity is important because many of the chemicals potentially present at the site (e.g., metals, chlorinated pesticides, PCBs) are persistent and can accumulate in sediments, potentially resulting in high exposures for benthic (sediment-dwelling) organisms. Sediments in the Seaplane Lagoon and the Oakland Estuary were selected for evaluation because these areas are known to have received waste discharges from the NAS Alameda in the past and therefore, potentially represent the marine environments most impacted by activities at NAS Alameda. Chemical contamination and associated impacts would be expected to be less at other points in the Bay more distant from the suspected source areas of the areas of highest contamination.

Bioassays should be conducted using benthic species indigenous to the NAS Alameda areas. The toxic endpoints measured should represent both lethal (i.e., survival) and nonlethal (e.g., emergence from eggs, growth) responses

as both types of effects can have direct effects on the health of the benthic community. Sediment samples for the bioassays should be selected randomly from the grid point locations used to collect surface water and sediment samples from these areas. The number of bioassays conducted should be sufficient to permit a statistical evaluation of the results; depending on the variation of the measured endpoints, at least 8 sediment samples from each of the study areas should be selected and assayed. Bioassays using clean (uncontaminated) sediment are needed to provide controls against which the results of bioassays using lagoon and estuary sediments can be compared. Additional control tests using sediments from other areas of the Bay that are similar ecologically but distant from NAS Alameda would be useful for interpreting the nature of background contamination in the Bay in relationship to the chemical levels detected in NAS Alameda sediments.

Tissue Residue Analyses

Tissue residue analyses are recommended to evaluate if chemicals associated with the site are accumulating in the food chain. Several of the chemicals potentially present at the site, including PCBs, DDT and other chlorinated pesticides, and some metals are know to bioaccumulate in aquatic species to concentrations much higher than those in the surrounding water. For example, biocentration factors in the range of 100,000 to 450,000 have been reported for PCBs in fish. Animals feeding on aquatic life that has bioconcentrated chemicals could potentially receive high doses of chemicals in food even if concentrations in the surrounding waters are low or undetectable. The species at greatest risk from such food-chain exposure are those located near the top of the food chain. Of particular concern at the NAS Alameda site are California least terns and brown pelicans. Both species feed on fish from the Seaplane Lagoon and adjacent waters, areas potentially impacted by past releases of chemicals from NAS Alameda. Further, both species are classified as endangered by the state and federal government and consequently, any toxic effects in the populations at NAS Alameda could impair the recovery of the species.

Therefore, it is recommended that fish be collected from the Seaplane Lagoon and analyzed for organochlorine pesticides, PCBs, metals, and other chemicals which have high bioconcentration potential. Analysis for other contaminants is probably not necessary since they are unlikely to bioaccumulate to a significant degree; however, a complete analysis for all priority pollutants in addition to the chemicals listed above would provide more definitive information on food-chain accumulation at NAS Alameda. The fish species collected should be those that compose the diet of California least terns and pelicans (e.g., jack smelt, top smelt, anchovy). Because fish are mobile, tissue residue levels will reflect exposure to chemicals throughout their range, as well as any chemical accumulation associated with NAS Alameda.

Therefore, fish tissue samples collected from other areas of the Bay are needed to distinguish background Bay contamination from chemical contamination associated with NAS Alameda. Background samples should be composed of the same species collected in the Seaplane Lagoon. Whole body chemical concentrations should be measured (for the human health assessment, concentrations in edible tissues [i.e., fillets] are needed). Chemical concentrations should be reported as dry weight. The lipid content of each sample should be measured as well as the percent moisture. Finally, the size and age of the fish also is needed.

Tissue residue levels also should be analyzed in benthic species in the two wetlands located on site. These areas are used as feeding areas by many species of shorebirds and ducks which feed primarily on benthic organisms. Again, background samples are needed with which to compare the samples from the on site wetlands. Also, whole-body chemical concentrations should be reported as dry-weight, and the percent lipid and moisture recorded.

7.1.7 Air

As discussed in the section on migration pathways, airborne chemicals may reach receptors by two routes: subsurface migration of chemical vapors or

landfill gases, and airborne transport of volatile or particle-bound chemicals from surface soils. The areas of potential concern for exposure to chemicals from these sources is likely to be different: subsurface vapors would be of concern primarily inside buildings, where vapors migrating from underground tanks or landfills could be trapped; chemicals volatilized from surface soils, or transported with dust particles would be of concern primarily outdoors. To evaluate risks from these sources, air samples should be taken both in locations of potential gas migration (as indicated by an organic vapor analyzer or other detector) and in locations downwind from unpaved areas of contaminated surface soil. Both particulates and vapors should be sampled in outdoor air. It is recommended that particle size distribution be determined for a subset of the samples, in order to evaluate the fraction of respirable particulates in the ambient air. Background air samples should be taken at locations upwind from known sources. At least one meteorological station will be required. Several air monitors should be placed at the fenceline and near the off-site receptors to the south and east of NAS Alameda.

7.2 RECEPTOR POPULATION DATA NEEDS

In addition to characterizing the occurrence and distribution of chemicals at the NAS Alameda site, the final public health evaluation will require identification of populations currently living and working in the vicinity of the site. Data are required on the numbers and demographic makeup of persons employed at the site, the average duration of employment or residence at the site, the location of the nearest residential areas, schools, and playgrounds, and the frequency of usage of areas at the NAS Alameda site for activities such as jogging, riding dirt bikes, picnicking, and fishing from the sea walls and piers.

8.0 CONCLUSIONS

This preliminary PHEE was conducted to identify potential human and environmental exposure pathways for detected and potential contaminants at the twenty study areas at NAS Alameda and to determine the data required to estimate the potential risks. Under current-use conditions, workers are most likely to be exposed to site contaminants via direct contact with contaminated soil in unpaved areas: the Fire Training Area, Building 389, and the Cans C-2 Area. Workers in the crawl space below Building 360 may also be exposed via direct contact. Individuals fishing in the Seaplane Lagoon may potentially be indirectly exposed to site contaminants via the consumption of contaminated fish. Both residents and workers may be exposed to airborne contaminants generated via volatilization from soil and airborne particulate generation. In areas with suspected subsurface hydrocarbon contamination (i.e., Buildings 459 and 547, Area 97, and Oil Refinery), residents with basements, and workers in crawl spaces may be exposed to hydrocarbon vapors. Additionally, recreational users of the Oakland Estuary and the picnic area may be exposed to site contaminants via inhalation and direct contact. At this time, no data exist to quantitatively evaluate potential human health risks that may be posed by contaminants at NAS Alameda. Based on the information available for this report, the Seaplane Lagoon and Area 97 with the associated subsurface contaminants appear to be of potential concern. Worker exposure via direct contact with surface contaminants is most likely in Building 360 where workers have access to the crawl space. As more data are available, other potential risks may be identified.

Future land-use may change and result in greater exposure to workers and potential future residents. As more data are available this will be evaluated further. At this time, conversion of NAS Alameda to residential or park property is considered unlikely unless NAS Alameda is closed.

Sites on NAS Alameda may pose risks to plants, terrestrial wildlife, and aquatic life. Due to the presence of a wetland on the former West Beach Landfill, plants and animals dependent on this area may be adversely affected

by landfill contaminants. The two landfills may potentially result in adverse effects on plants, on jackrabbits and other herbivorous mammals, and on shore-dwelling birds such as killdeer and plovers. Aquatic species and Bay-foraging birds may be exposed to contaminants present in the Seaplane Lagoon and Oakland Estuary.

In order to prepare a final Public Health and Environmental Evaluation for NAS Alameda, additional data are required. These data needs include surface soil samples to quantify direct contact risk; hydrogeological investigations to determine potential interconnectedness of aquifers beneath the site with off-site aquifers; surface water, sediment, and biota data from the Seaplane Lagoon; and meteorological investigations to identify potential downwind receptors.

9.0 REFERENCES

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987a. Draft Toxicological Profile for Chromium. Oak Ridge National Laboratory, October 1987.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1987b. Draft Toxicological Profile for Benzo[a]pyrene. October 1987.

AGENCY FOR TOXIC SUBSTANCE AND DISEASE REGISTRY (ATSDR). 1987c. Draft Toxicological Profile for Benzene; U.S. Public Health Service.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1988a. Draft Toxicological Profile for Methylene Chloride. U.S. Public Health Service.

AGENCY FOR TOXIC SUBSTANCES AND DISEASE REGISTRY (ATSDR). 1988b. Draft Toxicological Profile for PCBs. U.S. Public Health Service.

AHMED, F.E., HART, R.W., and LEWIS, J.J. 1977. Pesticide induced DNA damage and its repair in cultured human cells. Mutat. Res. 42:116-174.

ALBRO, P. and THOMAS, R. 1974. Intestinal absorption of hexachlorobenzene and hexachlorocyclohexane isomers in rats. Bull. Environ. Contam. Toxicol. 12: 289-294.

ALLIANCE TECHNOLOGIES CORPORATION (Alliance/WESTDIV). 1987. Summary of 20 sites at NAS-Alameda. Prepared for the Department of Navy, Western Division, Naval Facilities Engineering Command.

AMBROSE, A.M., LARSON, P.S., BORZELLECA, J.R., and HENNIGAR, G.R. 1976. Long-term toxicologic assessment of nickel in dogs and rats. J. Food Sci. Technol. 13:181-187.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1983. Threshold Limit Values for Chemical Substances and Physical Agents in the Workroom Environmental with Intended Changes for 1983-1984. ACGIH, Cincinnati, Ohio.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices. 5th ed. Cincinnati, Ohio.

AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (ACGIH). 1987-88. Documentation of the Threshold Limit Values and Biological Exposure Indices. Cincinnati, Ohio.

AMES, B.N., McCANN, J., and YAMASAKI, E. 1975. Methods for detecting carcinogens and mutagens with the salmonella/mammalian-microsome mutagenicity test. Mutat. Res. 31:347-364.

ANDERSEN K.J. et al. 1972. Evaluation of herbicides for possible mutagenic properties. J Agric Food Chem 20 (3): 649-56.

ANDERSON, P.H., MACHIN, A.F., and HERBERT, C.N. 1969. Blood cholinesterase activity as an index of acute toxicity of organophosphorus pesticides in sheep and cattle. Res. Vet. Sci. 10:29-33.

BAES, C.F., III, and SHARP, R.D. 1983. A proposal for estimation of soil teaching soil leaching and leaching constants for use in assessment models. J. Environ. Qual. 12: 17-28.

BAES, C.F., III, SHARP, R.D., SJOREEN, A.L., and SHOR, R.W. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides through Agriculture. Prepared for the U.S. Department of Energy. September 1984. ORNL-5786.

BAILEY, S.F. 1987. California Least Tern Foraging and Other Off-Colony Activities Around Alameda Naval Air Station During 1987. Department of Ornithology and Mammalogy. California Academy of Sciences. San Francisco, California.

BAKKE, J.E., and J.D. ROBBINS. 1968. Metabolism of atrazine and simazine by the goat and sheep. Abstr. Pap. 155th National Meeting Am. Chem. Soc. (A43).

BALYAEVA, A.P. 1967. The effects of antimony on reproduction. Gig. Truda Prof. Zabol. 11:32.

BANK, W.J. 1980. Thallium. In Spencer, P.S., and Schaumberg, H.H., eds. Experimental and Clinical Neurotoxicology. Williams and Wilkins, Baltimore. p. 571.

BANN, J.M., DeCINCO, T.J., EARL, N.W., and SUN, Y.P. 1956. The fate of aldrin and dieldrin in the animal body. Jour. Agri. Food Chem. 4:937.

BARDODEJ, Z., and BARDODEJOVA, E. 1970. Biotransformation of ethylbenzene, styrene, and alpha-methylstyrene in man. Am. Ind. Hyg. Assoc. J. 31:206-209.

BARKER, J.F., PATRICK, G.C., and MAJOR, D., 1987. Natural attenuation of aromatic hydrocarbons in a shallow sand aquifer. Ground Water Monitoring Rev., Winter 1987. pp. 64-71.

BARLOW, S.M., and SULLIVAN, F.M. 1982. Hazards of Industrial Chemicals. Evaluation of Animal and Human Data. Academic Press, pp. 119-125.

BARNES, D.W., SANDERS, V.M., WHITE, K.L., Jr., et al. 1985. Toxicology of trans-1,2-dichloroethylene in the mouse. Drug Chem. Toxicol. 8:373-392.

BARRIO-LAGE, G., PARSONS, F.Z., NASSAR, R.S., and LORENZO, P.A. 1986. Sequential dehalogenation of chlorinated ethenes. Environ. Sci. Technol. 20(1):96-99.

BERTRAND, R. and PEZERAT, H. 1980. Fibrous Glass: Carcinogenicity and dimensional characteristics. In Wagner, J.C. and Davis, W. eds. Effect Biologiques des Fibres Minerales, W.H.O., Lyons, France. pp. 901-911.

BIRNBAUM, L. 1986. Personal Communication. National Institute for Environmental Health Sciences. April, 1986.

BLUMBERG, H. and CAREY, T.N. 1934. Argyremia: Detection of unsuspected and obscure argyria by the spectrographic demonstration of high blood silver. J. Am. Med. Assoc. 103:1521-1524.

BONSE, G., URBAN, T., MONTESSANO, R., AND TOMATIS, L. 1975. Chemical reactivity, metabolic oxirane formation and biological reactivity of chlorinated ethylenes in the isolated perfused rat liver preparation. Biochem. Pharmacol. 24:1829-1834.

BOUTWELL, R.K., and BOSCH, D.K. 1959. The tumor-producing action of phenol and related compounds for mouse skin. Cancer Res. 19:413.

BROWN, D.S., CALTON, R.E., AND MULKEY, L.A. 1986. Development of land disposal decisions for metals. Draft. Assessment Branch. Environmental Research Laboratory, US-EPA, Athens, Georgia.

BRUCE, R.B., HOWARD, J.W., and ELSEA, J.R. 1955. Toxicity of o,o-diethyl o-(2-isoprophyl-6-methyl-4-pyrimidyl)phosphorothioate (diazinon). J. Agric. Food Chem. 3:1017-1021.

BRUCKNER, J.V., MACKENZIE, W.F., MURALIDHARA, S., et al. 1986. Oral toxicity of carbon tetrachloride: acute, subacute and chronic studies in rats. Fund. Appl. Toxicol. 6(1):16-34.

BUBEN, J.A., and O'FLAHERTY, E.J. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose-effect study. Toxico. Appl. Pharmacol. 78:105-122.

CABRAL, J.R.P., HALL, R.K., ROSSI, L., BRONKZYK, S.A., and SHUBIK, K.P. 1982. Effects of long-term intake of DDT on rats. Tumori. 68:11-17 (As cited in EPA, 1988a).

CAIN, B.W., SILEO, L., FRANSON, J.C., and MOORE, J. 1983. Effects of dietary cadmium on Mallard ducklings. Environ. Res. 32:286-297.

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (DHS). 1986. The California Site Mitigation Decision Tree Manual. Toxic Substances Control Division, Alternative Technology and Policy Development Section. May 1986.

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (DHS). 1987. The Development of Applied Action Levels for Soil Contact: A Scenario for the Exposure of Humans to Soil in a Residential Setting. Final Draft. Prepared by the Toxic Substances Control Division.

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (DHS). 1988a. Notice of Remedial Action Order, Health and Safety Code Section (s) 205, 206, 25220, 25355.5 (a) (1) (B), Docket No. HSA 88/89-051, letter dated June 30.

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (DHS). 1988b. Personal Communication with John Christopher. October 27, 1988.

CALIFORNIA DEPARTMENT OF HEALTH SERVICES (DHS). 1988c. List of applied action levels for substances in water, air and soil. December, 1988.

CANONIE ENVIRONMENTAL SERVICES, CORP. (Canonie/WESTDIV). 1988a. Sampling Plan, Remedial Investigation/Feasibility Study, Naval Air Station Alameda, Alameda, California, Volume 1. Prepared for Department of the Navy, Western Division, Naval Facilities Engineering Command, August.

CANONIE ENVIRONMENTAL SERVICES, CORP. (Canonie/WESTDIV). 1988b. Draft Sampling Plan, Remedial Investigation/Feasibility Study, Naval Air Station Alameda, Alameda, California, Volume 1a. Prepared for Department of Navy, Western Division, Naval Facilities Engineering Command, October.

CANONIE ENVIRONMENTAL SERVICES CORP. (Canonie/WESTDIV). 1988c. Quality Assurance Project Plan (QAPP), Quality Assurance/Quality Control Plan (QA/QC), Remedial Investigation/Feasibility Study, Naval Air Station Alameda, Alameda, California, Volume 3. Prepared for Department of the Navy, Western Division, Naval Facilities Engineering Command, August.

CANONIE ENVIRONMENTAL SERVICES CORP. (Canonie/WESTDIV). 1988d. Air Sampling Plan, Remedial Investigation/Feasibility Study, Naval Air Station Alameda, Alameda, California, Volume 1b. Prepared for Department of the Navy, Western Division, Naval Facilities Engineering Command, August.

CAREY, A.E., GOWEN, J.A., FOREHAND, T.J., TAI, H., and WIERSMA, G.B. 1980. Heavy metal concentrations in soils of five United States cities: 1972 urban monitoring program. Pestic. Monit. J. 13:150-154.

CARPENTER, C.P., WEIL, C.S., and SMTYH, H.F. 1953. Chronic oral toxicity of di(2-ethylhexyl)phthalate for rats, guinea pigs, and dogs. Arch. Indust. Hyg. Occup. Med. 8:219-226.

CATES, RANDY. 1988. Personal Communication.

CAVENDER, F.L., CASEY, H.W., SALEM, H., SWENBERG, J.A., and GARALLA, E.J. 1983. A 90-day vapor inhalation toxicity study of methyl ethyl ketone. Fund. Appl. Toxicol. 3:264-270.

CEREY, K., IZAKAVOC, V., and RUTTKAY-NEDECKA, J. 1973. Effects of heptachlor on dominant lethality and bone marrow in rats. (Abstract No. 10). Mutat. Res. 21:26.

CERNA, M., and KYPENOVA, H. 1977. Mutagenic activity of chloroethylenes analyses by screening system tests. Mutat. Res. 46: 214-215.

CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY (CIIT) 1980. A twenty-four month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. Executive Summary and Data Tables. October 15, 1980.

CHEMICAL INDUSTRY INSTITUTE OF TOXICOLOGY (CIIT). 1980. A Twenty-four Month Inhalation Toxicology Study in Fischer 344 Rats Exposed to Atmospheric Toluene. Executive Summary and Data Tables. October 15, 1980.

CHEN, C., CHUANG, Y., YOU, S., LIN, T., and WU, H. 1986. A retrospective study on malignant neoplasms of bladder, lung and liver in blackfoot disease endemic area in Taiwan. Brit. J. Cancer 53:399-405.

CHERNOFF, N., KAVLOCK, R.J., HANISCH, R.C.,. 1979. Perinatal toxicity of endrin in rodents. I. Fetotoxic effects of prenatal exposure in hamsters. Toxicology 13:155-165 (As cited in EPA 1985).

CHEVRON. 1985. Material Safety Data Sheet, Gulf bunker C fuel oil, No. AP020.

CHIN, B.H., McKELVEY, J.A., TYLER, T.R., CALISTI, L.J., KOZBELT, S.J., and SULLIVAN, L.J. 1980. Absorption, distribution and excretion of ethylbenzene, ethylcyclohexane and methyl ethylbenzene isomers in rats. Bull. Environ. Contam. Toxicol. 24:477-483.

CLAYTON, G.D., and CLAYTON, F.E., eds. 1984. Patty's Industrial Hygiene and Toxicology. 3rd. ed. John Wiley and Sons, New York. p. 1916.

CLEMENT ASSOCIATES. 1988. Multipathway Health Risk Assessment Input Parameters Guidance Document. June 1988.

COLLINS, L.D. 1987. California Least Tern Nesting Season at the Alameda Naval Air Station.

CONTE, F.S., and PARKER, J.C. 1975. Trans Am Fish Soc. 104:93-9 1975 (as cited in HSDB, 1988).

CORPS OF ENGINEERS (COE). 1985. Memo to Code 1142 From Code 243. Subject: Trip Report - status of wetlands at Naval Air Station, Alameda, July 22, 1985.

COTTON, F.A., and WILKINSON, G. 1982. Advanced Inorganic Chemistry. 3rd ed. John Wiley and Sons, New York.

COULSON, E.J., REMINGTON, R.E., and LYNCH, K.M. 1935. Metabolism in the rat of the naturally occuring arsenic of shrimp as compared with arsenic trioxide. J. Nutr. 10:255-276.

COUNCIL FOR AGRICULTURAL SCIENCE and TECHNOLOGY (CAST). 1976. Farm Chem., 138(13), 36(1975); Rep.No.59, September 15, 1976. As cited in Deichmann 1981.

COX, D.P. and ALEXANDER, M. 1973. Effect of Phosphate and Other Anions on Trimethylarsine Formation by Candida Humicola. Appl. Microb. 25(3):408.

COX, D.P. 1975. Methylation of Arsenic, IN: Arsenical Pesticides, ACS Symposium Series, R.F. Gould, ed.

CRANMER, J.S, AVERY, D.L., GRADY, R.R., and KITAY, J.I. 1978. Postnatal endocrine dysfunction resulting from prenatal exposure to carbofuran, diazinon, or chlordane. J. Environ. Pathol. Toxicol. 2:357-369.

CRECELIUS, E.A., JOHNSON, C.J., and HOFER, G.C. 1974. Contamination of soils near a copper smelter by arsenic, antimony and lead. Water Air Soil Pollut., 3:337.

CRECELIUS, E.A., BOTHNER, M.H. AND CARPENTER, R. 1975. Geochemistries of arsenic, antimony, and related elements in sediments of Puget Sound. Environ. Sci. Technol. 9(4):325-333.

CRONKITE, E.P., DREW, R.T., INOUE, T., and BULLIS, J.E. 1985. Benzene hematotoxicity and leukemogenesis. Am J Ind Med p. 133.

DAVIES, D.B., and HOLUB, B.J. 1980. Toxicological evaluation of dietary diazinon in the rat. Arch. Environ. Contam. Toxicol. 9:637-650.

DAVIS, G.M., and LEWIS, I. 1956. Outbreak of food poisoning from bread made of chemically contaminated flour. Br. Med. J. 11:393-398.

DE PROSPERO, J.R. 1972. Acute oral toxicity in rats: diazinon MG8. Affiliated Medical Research, Princeton, New Jersey for Geigy Agricultural Chemicals. MRID 00034096.

DEAN, R.B. 1981. Use of lognormal statistics in environmental monitoring. In: Chemicals and Water Use. Vol. I. Cooper, W.J., ed. Ann Arbor Science, Ann Arbor, MI.

DEICHMANN, W.B., MACDONALD, W.E., BLUM, E., et al. 1970. Tumorigenicity of aldrin, dieldrin, and endrin in the albino rat. Ind. Med. 39:426-434.

DEICHMANN, W.B. 1981. Halogenated cyclic hydrocarbons. In Patty's Industrial Hygiene and Toxicology. 3rd edition. John Wiley and Sons, New York. pp.3740-3749.

DEICHMANN, W.R. 1972. Toxicology of DDT and related chlorinated hydrocarbon pesticides. J. Occup. Med. 14:285-292.

DELLA PORTA, G.B., TERRACINI, B., and SHUBIK, K.P. 1961. Induction with carbon tetrachloride of liver cell carcinomas in hamsters. J. Natl. Cancer Inst. 26:855-863.

DIERCXSENS, P., deWECK, D., BORSINGER, W., ROSSET, B., and TARRADELLAS, J. 1985. Earthworm contamination by PCBs and heavy metals. Chemosphere. 14: 511-522.

DILLEY, J.V. 1977. Toxic evaluation of inhaled chlorobenzene. NIOSH, DHEW. Cincinatti, OH. Contract 210-76-0126.

DIPALMA, J.R., (ed.) 1971. Noxious gases and vapors: I. Carbon monoxide, cyanides, methemoglobin, and sulfhemoglobin. In Drill's Pharmacology in Medicine. McGraw-Hill Book Co., New York. Pp. 1189-1205.

DOW CHEMICAL CO. 1976. References and Literature Review Pertaining to Toxicological Properties of Phenol. Toxicol. Res. Lab. (unpublished manuscript) (As cited in EPA 1980j).

DOW CHEMICAL CO. 1983. Acc. No. 251473. Available from EPA (as cited in EPA 1988a).

DOWDY, R.J. and LARSON, W.E. 1975. The availability of sludge-borne metals to various vegetable crops. J. Environ. Qual. 4:278-282.

DRUET, P., DRUET, E., POTDEVIN, F., and SAPIN, C. 1978. Immune type glomerulonephritis induced by $HgCl_2$ in the Brown-Norway rat. Ann. Immunol. 129:777-792.

DSHUROV, A. 1979. Histological changes in organs of sheep in chronic simazine poisoning. Zentralbl. Veterinaermed. Reihe A. 26:44-54. (In German with English abstract).

E.I. duPONT de NEMOURS and Co., Inc. 1964. MRID. No. 00017763, 00091192.

EARL, F.L., MELUEGER, RINWALL, J.E., BIERBOWER, G.W., and CURTIS, J.M. 1968. Diazinon toxicity—comparative studies in dogs and miniature swine. Toxicol. Appl. Pharmacol. 12:287.

EAST, B.W., BODDY, K., WILLIAMS, E.D., MACINTYRE, D. and McLAY, A.L.C. 1980. Silver retention, total body silver and tissue silver concentrations in argyria associated with exposure to an anti-smoking remedy containing silver acetate. Clin. Exp. Dermatol. 5:305-311.

ECOLOGY AND ENVIRONMENT (E & E/WESTDIV). 1983. Initial Assessment Study, Naval Air Station, Alameda, California. Prepared for the Department of the Navy, Western Division, Naval Facilities Engineering Command. Contract No. N62474-82-C-8272.

EDWARDS, J., HESTON, W.E., and DALTON, H.A. 1942. Induction of carbon tetrachloride hepatoma in strain L mice. J. Natl. Cancer Inst. 3:297-301.

EDWARDS, N.T. 1983. Polycyclic Aromatic Hydrocarbons (PAHs) in the Terrestrial Environment - A review. J. Env. Qual. 12:427-441.

EISENBUD, M., and LISSON, J. 1983. Epidemiological aspects of beryllium-induced nonmalignant lung disease: A 20-year update. JOM J. Occop. Med. 25:198.

EISENREICH, S.J., LOONEY, B.B., THORTON, J.D. 1981. Airborne organic contaminants in the great Lakes ecosystem. Environ. Sci. Technol., 15:30-38.

EISLER, R. 1985. Cadmium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service. Biol. Report 85(1.2).

EISLER, R. 1986. Chromium Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service. Biol. Report 85(1.6).

EISLER, R. 1987a. Mercury Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service. Biol. Report 85(1.10).

EISLER, R. 1987b. Polycyclic Aromatic Hydrocarbons Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service. Biol. Report 85(1.11).

EISLER, R. 1988a. Arsenic Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service Biol. Report. 85(1.12).

EISLER, R. 1988b. Lead Hazards to Fish, Wildlife, and Invertebrates: A Synoptic Review. U.S. Fish and Wildlife Service Biol. Report. 85(1.14).

ENVIRONMENTAL ASSESSMENT ENGINEERING (EAE/WESTDIV). 1974. Preliminary Submission Candidate Environmental Impact Statement for Dredge Spoil Disposal Operations at the Naval Air Station, Alameda, California (As cited in E & E/WESTDIV, 1983).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1975. Initial Scientific and Minieconomic review of Monuron. Office of Pesticide Programs Criteria and Evaluation Division. EPA-54011-75-028.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1976. Interim Primary Drinking Water Regulations. Federal Register 40:34324 July 9.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1978. Occupational Health Guidelines for Chlordane, Lindane, Malathion, and Warfarin. Chemical, Physical and Biological Properties of Compounds Present at Hazardous Waste Sites. Vols. I, II, and III. Office of Waste Programs Enforcement. Office of Solid Waste and Emergency Response. September, 1978.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1979. Water-Related Environmental Fate of 129 Priority Pollutants. Washington, D.C. December 1979. EPA 440/4-79-029.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980a. Ambient Water Quality Criteria for Dichloroethylenes. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 440/5-80-041.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980b. Ambient Water Quality Criteria for Endosulfan. Office of Water Regulations and Standards. Washington, D.C. October 1980. EPA 440/5-80-046.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980c. Ambient Water Quality Criteria Document for DDT. Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-038.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980d. Ambient Water Quality Criteria for Antimony. Office of Water Regulations and Standards, Washington, D.C. EPA 440/5-80-020.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980e. Ambient Water Quality Criteria for Chromium. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-035.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980f. Ambient Water Quality Criteria for Mercury. Office of Water Regulations and Standards. Washington, D.C. EPA 440/5-80-053.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980g. Ambient Water Quality Criteria for Polychlorinated Biphenyls. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. (EPA/440/5-80-068). NTIS PB 81-117798.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980h. Ambient Water Quality Criteria for 2,4-dimethylphenol. Office of Water Regulations and Standards, Criteria and Standards Division. Washington D.C. October 1980. EPA 440/5-80-028.

ENVIRONMENTAL PROTECTION AGENCY (EPA) 1980i. Ambient Water Quality Criteria for Phthalate Esters. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 40/5-80-067.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980j. Ambient Water Quality Criteria for Phenol. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA/440/5-80-066.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1980k. Ambient Water Criteria for Silver. Office of Water, Washington, D.C. EPA 440/5-80-071. PB1-117822.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 19801. Ambient Water Quality Criteria for Selenium. Office of Water Regulations and Standards, Criteria and Standards Division, Washington, D.C. October 1980. EPA 400/5-80-070.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984a. Health Assessment Document for Carbon Tetrachloride. Environmental Criteria and Assessment Office. Cincinnati, OH September. EPA-600/8-82-001F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984b. Health Effects Assessment for cis-1,2-Dichloroethylene. Office of Emergency and Remedial Response. Cincinnati, OH EPA 540/1-86-015.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984c. Health Effects Assessment for Vinyl Chloride. Environmental Criteria and Assessment Office, Cincinnati, OH September 1984. EPA 540/1-86-036.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984d. Health Effects Assessment for Acetone. Environmental Criteria and Assessment Office, Cincinnati, OH EPA 540/1-86-016.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984e. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons (PAHs). Environmental Criteria and Assessment Office, Cincinnati, OH September 1984. EPA 540/1-86-013.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984f. Health Effects Assessment for Naphthalene. Environmental Criteria and Assessment Office, Cincinnati, OH September 1984. EPA 540/1-86-014.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984g. Health Effects Assessment Document for DDT. Office of Emergency and Remedial Response, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984h. Health Effects Assessment for Lindane. Office of Emergency and Remedial Response. Washington, D.C. September 1984. EPA 540/1-86-056.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984i. Health Effects Assessment for Barium. Environmental Criteria and Assessment Office, Cincinnati, OH September 1984. EPA 540/1-86-021.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984j. Health Assessment Document for Chromium. Environmental Criteria and Assessment Office, Research Triangle Park, N.C. EPA 600/8-83-014F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984k. Health Effects Assessment for Iron. Environmental Criteria and Assessment Office, September 1984. Cincinnati, OH.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 19841. Health Effects Assessment for Lead. Environmental Criteria and Assessment Office, Cincinnati, OH EPA/540/1-86-055.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984m. Health Effects Assessment for Mercury. Environmental Criteria and Assessment Office. Cincinnati, OH EPA 540/1-86-042.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984n. Health Effects Assessment for Polychlorinated Biphenyls. Environmental Criteria and Assessment Office, Cincinnati, OH September 1984. EPA 540/1-86-004.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984o. Health Effects Assessment for Zinc (and Compounds). Office of Emergency and Remedial Response, Washington, D.C. September 1984. EPA 540/1-86-048.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984p. Health Effects Assessment for Benzo[a]pyrene. Environmental Criteria and Assessment Office, Cincinnati, OH September 1984. EPA 540/1-86-022.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984q. Health Assessment Document for Inorganic Arsenic. Office of Health and Environmental Assessment, Washington, D.C. March 1984. EPA 600/8-83-021F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984r. Health Effects Assessment for Arsenic. Environmental Criteria and Assessment Office. Cincinnati, OH. September 1984. EPA 540/1-86-037.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984s. Proposed Guidelines for Carcinogen Risk Assessment; request for comments. November 23, 1984. Fed. Reg. 49: 46294-46301.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984t. Health Effects Assessment for Phenol. Environmental Criteria and Assessment Office, Cincinnati, OH September 1984. EPA 540/1-86-007.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984u. Health Effects Assessment for 1,1,1-Trichloroethane. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, OH September 1984. ECAO-CIN-H005.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984v. Health Assessment Document for Manganese. Environmental Criteria and Assessment Office, Environmental Protection Agency, Cincinnati, OH. Final Report. August 1984. EPA-600/8-83-013F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984w. Health Effects Assessment for Chlorobenzene. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-86-040.

ENVIRONMENTAL PROTECTION AGENCY (EPA) 1984x. Health Effects Assessment for Xylene. Environmental Criteria and Assessment Office, Cincinnati, OH EPA 540/1-86-006.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984y. Health Effects Assessment for Selenium. Environmental Criteria and Assessment Office, Cincinnati, OH September 1984. EPA 540/1-86-058.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1984z. Proposed Guidelines for Carcinogen Risk Assessment; request for comments. November 23, 1984. Fed. Reg. 49: 46294-46301.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985a. Drinking Water Criteria Document for Benzene. Final Draft. April 1985. Office of Drinking Water, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985b. Drinking Water Criteria Document for Toluene. Final Draft. Office of Drinking Water, Washington, D.C. March 1985.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985c. Drinking Water Criteria Document for Carbon Tetrachloride. Final Draft. January. Office of Drinking Water. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA) 1985d. Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Office of Health and Environmental Assessment, Washington, D.C. July 1985. EPA 600/8-82-005F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985e. Drinking Water Criteria Document for Tetrachloroethylene. Office of Drinking Water, Criteria and Standards Division. April 1985. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985f. Drinking Water Criteria Document on Vinyl Chloride. Final Draft. Office of Drinking Water. January 1985. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985g. Drinking Water Criteria Document for Endrin. Environmental Criteria and Assessment Office, Cincinnati, OH EPA-600/X-84-176.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985h. Draft Barium Health Advisory. Office of Drinking Water. September 30, 1985. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985i. Drinking Water Criteria Document for Lindane. Final Draft. Environmental Criteria and Assessment Office. March 1984. ECAO-CIN-402.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985j. Drinking Water Criteria Document for Cadmium. Final Draft. Office of Drinking Water, Washington, D.C. April 1985. PB86-117934.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985k. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium. Addendum to the Health Assessment Document for Cadmium (May 1981; EPA/600/8-81/023). Office of Health and Environmental Assessment, June 1985. Washington, D.C. EPA 600/8-83-025F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 19851. Drinking Water Criteria Document for Cyanide. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, OH EPA 600/X-84-192-1. PB86-117793.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985m. Health Effects Criteria Document on Polychlorinated Biphenyls. Final Draft. Office of Drinking Water, Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985n. National Primary Drinking Water Regulations; Synthetic Organic Chemicals, Inorganic Chemicals and Microorganisms. Fed. Reg. 50:46937-47025 (November 13, 1985).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985o. Health Assessment Document for Dichloromethane. Office of Health and Environmental Assessment, February 1985. Washington, D.C. EPA/600/8-82004F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985p. Addendum to the Health Assessment Document for Dichloromethane. Office of Health and Environmental Assessment, Washington, D.C. September 1985. EPA/600/8-82-004F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985q. Drinking Water Criteria Document for Heptachlor, Heptachlor Epoxide, and Chlordane. Final Draft. March, 1985. PB86-117991 EPA 600/x.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985r. Drinking Water Criteria Document for Selenium. Final Draft. Office of Drinking Water, Washington, D.C. Contract No. 68-01-6750. NTIS Publ. No. PB86-118098.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985s. Drinking Water Criteria for 1,1,1-Trichloroethane. Draft. Office of Drinking Water, Criteria and Standards Division. Washington, D.C. January, 1985.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985t. Health Assessment Document for Trichloroethylene. Environmental Criteria and Assessment Office. Research Triangle Park, N.C. EPA 600/8-82/006F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985u. Drinking Water Criteria Document for Copper. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, OH March 1985. EPA-600/X-84-190-1. NTIS publ. No. PB86-118239.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985v. Draft Health Advisory for Lead. Office of Drinking Water. September 30, 1985. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985w. Health Assessment Document for Chlorinated Benzenes. Final Report. Office of Health and Environmental Assessment, Washington, D.C. EPA/600/8-84/015F.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985x. Water Quality Criteria; Notice of Final Ambient Water Quality Criteria Documents. July 29, 1985. Fed. Reg. 50:30784-30796.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985y. Drinking Water Criteria Document for Silver. Environmental Criteria and Assessment Office, Cincinnati, OH PB 86-118288.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985z. Drinking Water Criteria Document for 2,4-dichlorophenoxyacetic acid (2,4-D). Final draft. March, 1985. Office of Drinking Water.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985aa. Carcinogenicity Risk Assessment for Chlordane and Heptachlor/Heptachlor Epoxide, Draft p.3-3. EPA Contract No. 68-02-4131 (as cited in HSDB, 1988).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1985ab. Drinking Water Criteria Document for Asbestos. Prepared by the Office of Health and Environmental Assessment, ECAO, Cincinnati, OH.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986a. Evaluation of the Potential Carcinogenicity of Benzene (71-43-2). Prepared by Carcinogen Assessment Group for Office of Emergency and Remedial Response and Office of Solid Waste and Emergency Response. Washington, D.C. OHEA-C-073-29.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986b. Ninety-Day Gavage Study in Albino Rats Using Acetone. Office of Solid Waste, Washington, D.C. (As cited in EPA 1988a.)

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986c. Health Assessment Document for Beryllium. Review Draft. Office of Health and Environmental Assessment, Washington, D.C. April 1986. EPA 600/8-84-026B.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986d. Health Assessment Document for Nickel and Nickel Compounds. Office of Health and Environmental Assessment, Research Triangle Park, N.C. EPA 600/8-83-012FF.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986e. (90-day) Toxicity of Thallium (I) Sulfate (CAS No. 7446-18-6) in Sprague Dawley Rats. Final Report. Prepared for the Office of Solid Waste, US EPA Washington, DC. Project No. 8702-1 (18).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986f. Superfund Public Health Evaluation Manual. Office of Emergency and Remedial Response. EPA/540/1-86/060.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986g. Drinking Water Criteria Document for Phthalic Acid Esters (PAEs). Draft document dated August 1986, with corrections as received on March 7, 1988. Environmental Criteria and Assessment Office, Cincinnati, OH

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986h. Airborne Asbestos Health Assessment Update. Office of Research and Development, Research Triangle Park, North Carolina. June 1986. EPA 600/8-84-003f.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986i. Guidelines for Carcinogen Risk Assessment. September 24, 1986. Fed. Reg. 51:33992-34003.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986j. Pesticide Fact Sheet No. 94. 2,4-D. July, 1986. Office of Pesticides and Toxic Substances, Office of Pesticide Programs.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986k. Guidelines for the Health Risk Assessment of Chemical Mixtures. Fed. Reg. Vol. 51:34014-34025.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1986l. Health and Environmental Effects Profile for Naphthalene. Environmental Criteria and Assessment Office, Cincinnati, OH

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987b. Memo from Sandra Lee (Program Manager, Regional Support Team, Toxics Integration Branch) to Addressees. Subject: Updated Reference Dose and Cancer Potency Numbers for Use in Risk Assessments. Office of Solid Waste and Emergency Response. Washington, D.C. October 1987. 19 pp. (Sandra Lee: (202) 382-4307).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987c. Health Advisory for cis-1,2-Dichloroethylene. Office of Drinking Water, March 31, 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987d. Draft Health Advisory for Simazine. Office of Drinking Water. August 1987. Washington, DC.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987e. Health Advisory for Diazinon. Draft. Office of Drinking Water. August 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987f. Draft Health Advisory for Mercury. Office of Drinking Water. September 30. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987g. Health Advisory for Nickel. Office for Drinking Water. March 31, 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987h. Health Advisory for Toluene. Office of Drinking Water. March 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987i. Health Advisory. Heptachlor and Heptachlor Epoxide. Office of Drinking Water. March 31, 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987j. Draft Health Advisory for Trichloroethylene. Office of Drinking Water. March 31, 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987k. Health Advisory for Ethylbenzene. Office of Drinking Water. March 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 19871. Health Advisory for 1,1,1-Trichloroethane. Office of Drinking Water, Criteria and Standards Division. March 31, 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987m. Health Advisory for Xylenes. Office of Drinking Water. March 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987n. Superfund Program: Interim Guidance on Compliance with Applicable or Relevant and Appropriate Requirements. August 27, 1987. Fed. Reg. 52:32496-32499.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987o. Drinking Water Criteria Document for Polychlorinated Biphenyls (PCBs). Final ECAO-CIN-414. (as cited in ATSDR 1988b).

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987p. Health Advisory for trans-1,2-Dichloroethylene. Office of Drinking Water, March 31, 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987q. Draft Health Advisory for Glyphosate. Office of Drinking Water, August 1987. Washington, D.C.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987r. Superfund Program: Interim Guidance on Compliance with Applicable or Relevant and Appropriate Requirements. August 27, 1987. Fed. Reg. 52:32496-32499.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1987s. Superfund Exposure Assessment Manual. Office of Emergency and Remedial Response.

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988a. Integrated Risk Information System (IRIS). Environmental Criteria and Assessment Office, Cincinnati, OH

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988b. June Quarterly Update, Health Effects Assessment Summary Table. Environmental Criteria and Assessment Office, June 1988. Cincinnati, OH

ENVIRONMENTAL PROTECTION AGENCY (EPA). 1988c. June Quarterly Update for HEA and HEED Chemicals. Memorandum from Chris DeRosa. July 15, 1988. Environmental Criteria and Assessment Office, Cincinnati, OH

EPSTEIN, S.S. 1975. The carcinogenicity of dieldrin. Part 1. (Review of: Davis, H.J., Hansen, W., and Fitzhugh, O.G. Pathology report on mice for aldrin, dieldrin, haptachlor or heptachlor epoxide for 2 years (ref. 2, p. 341) Sci. Total Environ. 4: 1-52.

EPSTEIN, S.S. 1976. Carcinogenicity of heptachlor and chlordane. Sci. Total Environ. 6:103-154.

ERM-West (ERM-West/WESTDIV). 1987. Tank Testing Study, Alameda Naval Air Station, Alameda, California, final report prepared for Western Division Naval Facilities Engineering Command. August. San Bruno, CA

FELDMAN, R.J., and MAIBACH, H.I. 1974. Percutaneous penetration of some pesticides and herbicides in man. Toxicol. Appl. Pharmacol. 28:126-132.

FERON, V.J., SPEEK, A.J., WILLIAMS, M.I., VAN BATTUM, D., and DE GROOT, A.F. 1975. Observations on the oral administration and toxicity of vinyl chloride in rats. Food. Cosmet. Toxicol. 13:633-638.

FERON, V.J., HENDRIKSEN, C.F.M., SPEEK, A.J., TIL, H.P., and SPIT, B.J. 1981. Lifespan oral toxicity study of vinyl chloride in rats. Food Cosmet. Toxicol. 19:317-333.

FITZHUGH, O.J., NELSON, A.A., and FRANLEY, J.P. 1950. J. Pharmacol. Exp. Ther., 100,59. (As cited in Deichmann 1981).

FITZHUGH, O., NELSON, A., LAUG, E., and KUNZE, F. 1950. Chronic oral toxicities of mercuric-phenyl and mercuric salts. Arch. Ind. Hyg. Occup. Med. 2:433-441.

FITZHUGH, O., NELSON, A.A., and QUAIFE, M.L. 1964. Chance oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2:551-562.

FOOD AND AGRICULTURAL ORGANIZATION /WORLD HEALTH ORGANIZATION (FAO/WHO). 1967. Evaluation of Some Pesticide Residues in Food. Geneva, Switzerland: FAO PL: CP/15, WHO/FOOD Add/67.32.

FOY, C.D., CHANEY, R.L. and WHITE, M.C. 1978. The physiology of metal toxicity in plants. Ann. Rev. Physiol. 29:511-566.

FREUNDT, K.J., LIEBALDT, G.P., and LIEBERWIRTH, E. 1977. Toxicity studies on trans-1,2-dichloroethylene. Toxicology 7:141-153.

FREUNDT, K.J., and MACHOLZ, J. 1978. Inhibition of mixed function oxidases in rat liver by trans- and cis-1,2-Dichloroethylene. Toxicology 10: 131-139.

FROST, R.R., and GRIFFIN, R.A. 1977. Effect on pH on adsorption of arsenic and selenium from landfill leachate by clay materials. Soil Sci. Soc. Am. J. 41:53-57.

FUKUDA, K., TAKEMOTO, K., and TSURUTA, H. 1983. Inhalation carcinogenicity of trichloroethylene in mice and rats. Ind. Health. 21:243-254.

FULLER, W.H. 1977. Movement of Selected Metals, Asbestos, and Cyanide in Soil; Applications to Waste Disposal Problems. April. Prepared for the Municipal Research Laboratory, EPA 600/2-77-020, NTIS PB-266 905.

FULLER, W.H. 1978. Investigation of Landfill Leachate Pollutant Attenuation by Soils, EPA/60012-78-158, NTIS-PB-286 995. Prepared for the Municipal Environmental Research Laboratory, Office of Research and Development. Cincinnati, OH: USEPA.

FURR, A.K., PARKINSON, T.F., GUTENMANN, W.H., PAKKALA, I.S. and LISK, D.J. 1978. Elemental content of vegetables, grains and forages field-grown on fly ash amended soil. J. Agric. Food Chem. 26:357-359.

FURST, A., and SCHLAUDER, M.C. 1977. Inactivity of two noble metals as carcinogens. J. Environ. Pathol. Toxicol. 1:51-57 (As cited in EPA 1985y).

GALLI, A., BRONZETTI, G., CORSI, C., del CARRATORE, R., NIERI, R., and PAOLINE, M. 1982b. (a) Studio in vivo. Attivita genetica dell' 1,2-Dichloroethylene. Boll. Soc. Ital. Biol. Sper. 58: 864-869.

GALLI, A., BRONZETTI, G., CORSI, C., del CARRATORE, R., NIERI, R., and PAOLINE, M. 1982a. Genetic activity of 1,2-Dichloroethylene. A. In Vitro studies. Boll. Soc. Ital. Biol. Sper. 58: 860-863 (Italian) (Abstract from NLM Toxline Database).

GARDINER, J. 1974. The chemistry of cadmium in natural waters: I. A study of cadmium complex formation using the cadmium specific-ion electrode. Water Res. 8:23-30.

GAUL, L.E. and STAUD, A.H. 1935. Clinical spectroscopy. Seventy cases of generalized argyrosis following organic and colloidal silver medication. J. Am. Med. Assoc. 104:1387-1390.

GERRITSE, K.G., VRIESMA, R., DALENBERG, J.W., and DeBOSS, H.P. 1982. Effect of sewage sludge on trace metal mobility in soils. J. Environ. Qual. 11: 359-364.

GIBSON, D.T., KOCH, J.R., and KALLIO, R.E. 1968. Oxidative degradation of aromatic hydrocarbons by microorganisms: I. Enzymatic formation of catechol from benzene. Biochemistry :653-2662.

GIBSON, J.E., and BECKER, B.A. 1970. Placental transfer, embryo toxicity, and teratogenicity of thallium sulfate in normal and potassium-deficient rats. Toxicol. Appl. Pharmacol. 16:120-132.

GILMAN, J.P.W. 1962. Metal carcinogenesis: II. A study on the carcinogenic activity of cobalt, copper, iron, and nickel compounds. Cancer Res. 22:158-165.

GLOVER, J.R. 1967. Selenium in human urine: A tentative maximum allowable concentration for industrial and rural populations. Ann. Occup. Hyg. 10:3-10.

GOCHFELD, M. and BURGH, J. 1988. Effects of lead on growth and feeding behavior of young common terms (Sterna hirunda). Arch. Env. Contam. Tox. 17:513-517.

GOOD, E.E. and WARE, G.W. 1969. Effects of insecticides on reproduction in the laboratory mouse. IV. Endrin and dieldrin. Toxicol. Appl. Pharmacol. (As cited in EPA 1985). 14:201-203.

GORE, R.C. et al. 1971. J Assoc Off Anal Chem 54: 1040-82 (As cited in HSDB, 1988).

GOSSELIN. CTCP 5TH ED. 1984 II-330 (as cited in HSDB, 1988).

GOYER, R.A. 1986. Toxic effects of metals. In Klaasen, C.D., Amdur, M.D., and Doull, J., (eds.) Casarett and Doull's Toxicology: The Basic Science of Poisons. 3rd ed. Macmillan Publishing Co., New York. Pp. 623-624.

GRANT. ITOX OF THE EYE 1974 , p. 605 (as cited in HSDB, 1988).

GRAY, L.E., Jr., KAVLOCK, R.J., CHERNOFF, N., GRAY, J.A., and McLAMB, J. 1981. Perinatal toxicity of endrin in rodents. III. Alterations of behavioral ontogeny. (As cited in EPA 1985g). Toxicology 21:187-202.

GREIM, H., BONSE, G., RADWAN, Z., REICHERT, D., and HENSCHLER, D. 1975. Mutagenicity in vitro and potential carcinogenicity of chlorinated ethylenes as a function of metabolic oxiran formation. Biochem. Pharmacol. (As cited in EPA 1980). 24: 2013-2017.

GRIFFIN, R.A. CHOU, S.F.J. 1981. Movement of PCBs and other persistent compounds through soil. Water Sci Technol; 13:1153-63.

GUPTA, P., CHANDRA, S., and SAXENA, D. 1978. Teratogenic and embryonic effects on endosulfan in rats. Acta Pharmacol. Toxicol. (As cited in EPA 1980). 42: 150-152.

GUY, R.D., and CHAKRABARTI, C.L. 1976. Studies of metal-organic interactions in model systems pertaining to natural waters. Can. J. Chem. 54:26002611.

HAMMOND, P.B., and BELILES, R.P. 1980. Metals. In: Casarett and Doull's Toxicology: The Basic Science of Poisons, 2nd Ed., Doull, J., Klaassen, C.D., and Amdur, M.O. (eds.), Macmillan Publishing Co., New York, Toronto, and London. pp. 421-428.

HANNINEN, H., ESKELININ, L., HUSMAN, K., and NURMINEEN, M. 1976. Behavioral effects of long-term exposure to a mixture of organic solvents. Scand. J. Work Environ. Health. (As cited in EPA 1987). 2:240-255.

HANSEN, W.H., QUAIFE, M.L., HABERMANN, R.T., and FITZHUGH, O.G. 1971. Chronic toxicity of 2,4-dichlorophenoxyacetic acid in rats and dogs. Toxicol. Appl. Pharmacol. 20(1):122-129.

HAPKE, H. 1968. Research into the toxicology of weedkiller simazine. Berl. Tieraerztl. Wochenschr. 81:301-303.

HAQUE, R. (ed.) 1970. Dynamics Exposure and Hazard Assessment of Toxic Chemicals. Ann Arbor Sci. Publ. Inc. Ann Arbor, MI.

HAQUE, R., and FREED, V.H. 1975. (eds) Environmental Dynamics of Pesticides. Plenum Press, NY

HARDIN, B.D., BOND, G.P., SIKOV, M.R., ANDREW, F.D., BELILES, R.P., and NIEMEIER, R.W. 1981. Testing of selected workplace chemicals for teratogenic potential. Scand. J. Work Environ. Health 7:66-75.

HARDING-LAWSON ASSOCIATES (Harding-Lawson Associates/WESTDIV). 1978. Final Submittal, Sanitary Landfill Site Study, Naval Air Station, Alameda, California, March 1. Report prepared for Commanding Officer, Western Division, Naval Facilities Engineering Command.

HARDING-LAWSON ASSOCIATES (Harding-Lawson Associates/WESTDIV). 1983. Confirmation Study, Sanitary Landfill, Naval Air Station, Alameda, California, October 19. Report prepared for Commanding Officer, Western Division, Naval Facilities Engineering Command.

HARDING-LAWSON ASSOCIATES (Harding-Lawson Associates/WESTDIV). 1988. Guidance for Conducting Remedial Investigations and Feasibility Studies Under CERCLA. Draft. March. OSWER Directive 9335.3-01.

HASELTINE, S.D., SILEO, L., HOFFMAN, D.J., and MULHERN, B.M. 1985. Effects of Chromium on Reproduction and Growth of Black Ducks. Manuscript in Preparation. (as cited in Eisler 1986).

HAYES. 1975. TOX OF PESTICIDES, p. 193 (as cited in HSDB, 1988).

HAYES, W.J., JR. 1963. Clinical Handbood on Economic Poisons. Emergency Information for Treating Poisoning. Public Health Service Publ. No. 476. U.S. Government Printing Office, Washington, D.C.

HAYES, W.J., JR. 1982. Pesticides Studied in Man. Williams and Williams, Baltimore. Pp. 385-389.

HAZARDOUS SUBSTANCES DATABASE [On Line] (HSDB), 1988.

HAZELETTE, J.R. 1984. Dietary hyperchloesterolemia and susceptibility to the pesticide diazinon. Diss. Abstr. Int. B. 44:2116.

HAZLETON, L.W. and HOLLAND, E.G. 1953. Toxicity of malathion. Summary of mammalian investigations. Arch. Ind. Hyg. Occup. Med., 8,399-405.

HAZELTON LABORATORIES. 1961. Two-year chronic feeding study in rats. CBI, Document No. 000525, MRID 0059211.

HAZELTON LABORATORIES. 1983. Document Accession Number 251473. U.S. EPA, Office of Pesticides Programs, Washington, D.C.

HEATH, J.C. 1960. The histogenesis of malignant tumors induced by cobalt in the rat. Br. J. Cancer 14:478-482.

HEATH, D.F., and VANDEKAR, M. 1964. Toxicity and metabolism of dieldrin in rats. Br. J. Ind. Med. 21:269-279.

HEINZ, G.H. and HASELTINE, S.D. 1981. Avoidance behavior of young black ducks treated with chromium. Toxicol. Lett. 8:307-310.

HEINZ, G.H. and HASELTINE, S.D. 1983. Altered avoidance behavior of young black ducks fed cadmium. Environ. Toxicol. Chem. 2:419-421.

HERBICIDE HANDBOOK. 1979. Fourth edition. P. 68 (as cited in HSDB, 1988).

HILL, W.R. and PILLSBURY, D.M. 1939. Argyria, the Pharmacology of Silver. Williams and Wilkins Co., Baltimore, Maryland.

HOESCHST AKTIENGESELLSCHAFT. 1984. Acc. Nos. 256127, 257727 (as cited in EPA 1987a).

HOFFMAN, D.J., FRANSON, J.C., PATTEE, O.H., BUNCK, C.M., and MURRAY, H.C. 1985. Biochemical and hematological effects of lead ingestion in nestling American kestrals ((Falco sparverius). Comp. Biochem. Physiol. 80c:431-439.

HONBA, C., REMADE, J. DUBOIS, D., and THOREZ, J. 1983. Factors affecting the concentrations of cadmium, zinc, copper and lead in the sediments of the Vesdie River. Water Res. 17: 1281-1286.

HOWARD, J.W. and HANZAL, R.F. 1985. Chronic toxicity for rats of food treated with hydrogen cyanide. J. Agric. Food Chem. 3:325-329.

HUANG, C.P., ELLIOTT, H.A., and ASHMEAN, R.M. 1977. Interfacial reactions and the fate of heavy metals in soil water systems. J. Water Pollut. Control 49: 745-756.

HUDAK, A., and UNGVARY, G. 1978. Embryotoxic effects of benzene and its methyl derivatives: toluene, xylene. Toxicology 11:55-63.

HUFF, B.B., (ed.) 1985. Physicians Desk Reference, 39th ed. Medical Economics, Co., Inc., Oradele, N.J. (as cited in EPA 1988a).

HUNTER, C.G., and ROBINSON, J. 1967. Pharmacodynamics of dieldrin (HEOD). I. Ingestion by humans for 18 months. Arch. Environ. Health 15:614-626.

HUNTER, C.G., ROBINSON, J., and ROBERTS, M. 1969. Pharmacodynamics of dieldrin (HEOD). Ingestion by human subjects for 18-24 months and post exposure for eight months. Arch. Environ. Health 18:12-21.

HYDRO-SEARCH, 1977. (Hydro-Search/Navy Public Works). Well Performance Evaluation Pan American Well, Naval Air Station, Alameda, California, Report of navy Contract No. NOO 228-77-M-M847, Report to Utilities Department, Navy Public Works Center, Oakland, California.

ICF INCORPORATED. 1986. Draft report. Development of soil: water distribution coefficients for LLM inorganic chemicals.

INGLE, L. 1952. Arch. Ind. Hyg. Occup. Med. 6,354 (as cited in Deichmann 1981).

INNES, J.R.M., 1968. Evaluation of Carcinogenic, Teratogenic, and Mutagenic Activities of Selected Pesticides and Industrial Chemicals. Volume 1: Carcinogenic Study. Bionetics Research Laboratories, Inc. Distributed by National Technical Information Service, Springfield, VA

INNES, J.R.M., ULLAND, B.M., VALERIO, M.G., PETRUCELLI, L., FISHBEIN, L., HART, E.R., PALLOTA, A.J., BATES, R.R., FALK, H.L., GART, J.J., KLEIN, M., MITCHELL, I., and PETERS, J. 1969. Bioassay of pesticides and industrial chemicals for tumorigenicity in mice: A preliminary note. J. Natl. Cancer Inst. 42:1101-1114.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1975. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Vol. 9: Some Aziridines, N-, S-, and O-Mustards, and Selenium. World Health Organization, Lyon, France. pp. 245-260.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1976. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Man. Vol. 12. Some Carbamates, Thiocarbamates and Carbazides. p. 171. World Health Organization. Lyon, France.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979a. Evaluation of the carcinogenic risk of chemicals to humans. Vinyl chloride, polyvinyl chloride and vinyl chloride. Vinyl acetate polymers. World Health Organization. Lyon, France: IARC; 19:377-401; 419-438.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1979b. IARC Mongraphs on the evaluation of the carcinogenic risks of chemicals to humans. Vol. 20: Some Halogented Hydrocarbons. World Health Organization. Lyon, France.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1980. Some Metals and Metallic Compounds. Vol. 23: IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. World Health Organization, Lyon, France. Pp. 143-204.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1982. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Volume 27: Some Aromatic Amines, Anthraquinones and Nitroso Compounds, and Inorganic Fluorides Used in Drinking-Water and Dental Preparations. World Health Organization, Lyon, France.

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER (IARC). 1986. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Halogenated Hydrocarbons and Pesticide Exposures. World Health Organization, Lyon, France pp. 357-406.

IRISH, D.D. 1963. Vinylidene chloride. In Patty, F.A., ed. Industrial Hygiene and Toxicology. 2nd ed. John Wiley and Sons, New York. Vol. II, pp. 1305-1309.

ITO, N., NAGASAKI, H., ARAI, M. et al. 1973. Histological and ultrastructural studies on the hepatocarcinogenicity of benzenehexachloride in mice. J. Natl. Cancer Inst. 51:817-826.

IVANKOVIC, S., and PREUSSMAN, R. 1975. Absence of toxic and carcinogenic effects after administration of high doses of chromic oxide pigment in subacute and long-term feeding experiments in rats. Fd. Cosmet. Toxicol. 13:347-351.

JAEGAR, R.J., SHONER, L.G., and COFFMAN, L.J. 1977. 1,1-Dichloroethylene hepatotoxicity: Proposed mechanism of action of distribution and binding of ^{14}C radioactivity following inhalation exposure in rats. Environ. Health Perspect. 21: 113-119.

JAMES, B.R. and BARTLETT, R.J. 1983. Behavior of chromium in soils: V. Fate of organically complexed Cr III added to soil. J. Environ. Qual. 5:169-172.

JENNE, E.A. 1968. Controls on Mn, Fe, Co, Ni, Cu, and Zn Concentrations in Soils and Water: The significant role of hydrous Mn and Fe oxides. In: Gould, R.F., (ed.). Trace Inorganics in Water: Advances in Chemistry 73. Washington, D.C., American Chemical Society.

JURY, W.A. 1985. J Environ Qual 12: 558-64 1983, (as cited in HSDB, 1988).

KABATA-PENDIAS, A., and PENDIAS, H. 1984. Trace Elements in Soils and Plants. CRC Press, Boca Raton, FL.

KANAZAWA, J. 1978. Bioconcentration ratio of diazinon by freshwater fish & snail; Bull. Environ. Contam. Toxicol. 20(5) 613 1978, (as cited in HSDB, 1988).

KANISAWA, M. and SCHROEDER, H.A. 1967. Life term studies on the effects of arsenic, germanium, tin and vanadium on spontaneous tumors in mice. Cancer Res. 27: 1192-1195.

KANISAWA, M. & SCHROEDER, H.A. 1969. Life term studies on the effect of trace elements on spontaneous tumors in mice and rats. CNRE 29:892-895.

KAO, J.K., PATTERSON, F.K., and HALL, J. 1985. Skin penetration and metabolism of topically applied chemicals in six mammalian species including man: An in vitro study with benzo[a]pyrene and testosterone. Toxicol. Appl. Pharmacol. 81:502-516 (As cited in ATSDR 1987).

KARNOFSKY, D.A., RIDGWAY, L.P., and PATTERSON, P.A. 1950. Production of achondroplasia in the chick embryo with thallium. Proc. Soc. Exp. Biol. Med. 73:255-259.

KEAMEY, P.C., and KAUFMAN, D.D. 1975. (eds). Herbicides Vo. 1. Marcel Dekker, Inc. NY

KELLY, P. 1988. Personal communication with Paul Kelly, California Department of Fish and Game. October 6, 1988.

KENNEDY ENGINEERS (Kennedy/WESTDIV). 1980. Subsurface Fuel Contamination Study for United States Navy. Prepared for Department of Navy, Western Division, Naval Facilities Engineering Command.

KHAN, MAQ et al. 1970. Ecological and health effects of the photolysis products of chlorinated hydrocarbon pesticides; In: Dynamics, Exposure and Hazard Assessment of Toxic Chemicals; Haque, R. (ed), Ann Arbor Sci. Publ Inc; Ann Arbor; Ch. 25: 393-415. (as cited in HSDB, 1988).

KHAN, S., NANDAN, D., and KHAN, N.N. 1982. The mobility of some heavy metals through Indian red soil. Environ. Poll. Ser. B., 4:119-125.

KHARKA, D.P., TUREKIAN, K.K., BERTINE, I.C.K. 1968. Stream supply of dissolved silver, molybdenum, antimony, selenium, chromium, cobalt, rubidium, and cesium to the oceans. Geochim. Cosmochim. Acta. 32:284-298.

KHERA, et al. 1979. Toxicol. Appl. Pharmacol. 48 (1): A33 (as cited in HSDB, 1988).

KIMBROUGH, R.D. and GAINES, T.B. 1968. Effect of organic phosphorus compounds and alkylating agents on the rat fetus. Arch. Environ. Health, 16,805-808.

KIMBROUGH, R.D., SQUIRE, R.A., LINDER, R.E., STRANDBERG, J.D., MONTALI, R.J. and BURSE, V.W. 1975. Induction of liver tumors in Sherman strain female rats by polychlorinated biphenyl Aroclor 1260. J. Natl. Cancer Inst. 55:1453.

KIMMERLE, G., and EBEN, A. 1973. Metabolism, excretion and toxicology of trichloroethylene after inhalation. 1. Experimental exposure on rats, Arch. Toxicol. 30:115.

KIMURA, E.T., EBERT, D.M., and DODGE, P.W. 1971. Acute toxicity and limits of solvent residue for sixteen organic solvents. Toxicol. Appl. Pharmacol. 19:699-704.

KJELLSTRAND, P., KANJE, M., MANSSON, L., BJERKAMO, M., MORTENSEN, I., LANKE, J., and HOLMQUIST, B. 1981. Trichloroethylene: Effects on body and organ weights in mice, rats, and gerbils. Toxicology; 21:105-115 (As cited in ATSDR 1988).

KLAASSEN, C.D., AMDUR, M.D., and DOULL, J., (eds.) 1986. Casarett and Doull's Toxicology. 3rd ed. Macmillan Publishing Co., New York.

KOLLER, L. 1979. Methylmercury toxicity, model no. 158. In: Jones, T.C., Hackel, D.B., and Migaki, G., (eds.) Handbook: Animal Models of Human Disease. Registry of Comparative Pathology, Armed Forces Institute of Pathology, Washington, D.C.

KORANSKY, W. and PORTIG, J. 1963. Arch. Exp. Pathol. Pharmakol., 244,564. (As cited in Deichmann 1981).

KORTE, N.E., SKOPP, J., FULLER, W.H., NIEVLA, E.E., and ALESII, B.A. 1976. Trace element movement in soils: Influence of soil physical and chemical properties. Soil Sci. 122: 350-525.

KRASAVAGE, W.J., O'DONOGHUE, J.L., and DIVINCENZO, G.D., 1982. Ketones, Chapter 56. In: Patty's Industrial Hygiene and Toxicology; Vol. IIB.

LABELLE, C.W., and BRIEGER, H. 1955. Vapor toxicity of a composite solvent and its principal components. Arch. Ind. Health 12:623-627.

LAGOY, P.K. 1987. Estimated Soil Ingestion Rates for Use in Risk Assessment. Risk Analysis 7:355-359.

LANDE, S.S., DURKIN, P.R., CHRISTOPHER, D.H., HOWARD, P.H., and SAXENA, J. 1976. Investigation of Selected Potential Environmental Contaminants: Ketonic Solvents. U.S. Environmental Protection Agency, Washington, D.C. EPA 560/2-76-003.

LAUG, E.P., NELSON, A.A., FITZHUGH, O.G., and KUNZE, F.M. 1950. Liver cell alteration and DDT storage in the rat of the rat induced by dietary levels of 1-50 ppm DDT. J. Pharmacol. Exp. Ther. 98:268-273.

LAXEN, D.H., DAVISON, W. and WOOL, C. 1984. Manganese chemistry in rivers and streams. Geochem. Cosmichm. Acta. 48:2107-2111.

LEIFER, A., BRINK, R.H., THOM, G.C., and PARTYMILLER, K.G. 1983. Environmental transport and transformation of polychlorinated biphenyls. EPA-560/5-83-025. Washington, D.C.: Office of Pest Tox Sub; 206 pp. NTIS No. PB84-1422579.

LEONARD, A., GERBER, G.B., JACQUET, P., and LAUWERYS, R.R 1984. Mutagenicity, carcinogenicity and teratogenicity of industrially used metals. In: Mutagenicity, Carcinogenicity and Teratogenicity of Industrial Pollutants. Kirsch-Volders, M. (eds.), Plenum Press, New York and London, pp. 59-126.

LITTLE, ARTHUR D., INC. 1976. Literature study of selected potential environmental contaminants; antimony and its compounds. pp. 5-15. 251-438. 147p.

LYMAN, W.J., REEHL, W.F., ROSENBLATT, D.H. 1982. Handbook of Chemical Property Estimation Methods. McGraw-Hill. New York.

MAAZIKA, I.K. 1968. Dimethylphenol (xylenol)isomers and thin standard contents in water bodies. Gig. Sanit 9:18.

MACKAY, D., and SHIU, W.Y. 1981. A critical review of Henry's law constants for chemicals of environmental interest. J. Phys. Chem. Ref. Data; 19:1175-1179.

MACKENZIE, R.D., BYERRUM, R.V., DECKER, C.F., HOPPERT, C.A., AND LONGHAM, F.L. 1958. Chronic toxicity studies II. Hexavalent and trivalent chromium administered in drinking water to rats. Arch. Ind. Health. 18:232-234.

MAGORIAN, T.R., WOOD, K.G., MICHALOVIC, J.G., PEK, S.L. and VAN LIER, M.M. 1974. Water Pollution by Thallium and Related Metals (NTIS PB 253 333, Springfield, Virginia) (cited in EPA, 1979).

MAHAFFEY, K.R. 1978. Environmental exposure to lead. In: Nriagu, J.O. (ed.). The Biochemistry of Lead in the Environment: Part B. Biological Effects. Elsevier/North-Holland Biomedical Press, New York. 397 pages.

MAIER-BODE, H. 1968. Properties, effect, residues and analytics of the insecticide endosulfan. Res. Rev. 22: 1-44.

MALTONI, C., CONTI, B., COTTI, G., and BELPOGGI, F. 1985. Experimental studies on benzene carcinogenicity at the Bologna Institute of Oncology: Current results and ongoing research. Am. J. Ind. Med. 7:415-446.

MANCUSO, T.F. 1975. International Conference on Heavy Metals in the Environment. Toronto, Canada.

MARKARJAN, D.S. 1966. Cytogenetic effect of some chlororganic insecticides on the nuclei of mouse bone-marrow cells. Genetika 1:132-137.

MAUTINO, M. and Bell, J.U. 1987. Hematological evaluation of lead intoxication in Mallards. Bull. Environ. Contam. Toxicol. 38:78-85.

MAXFIELD, D.J., RODRIGUEZ, M., BUETTNER, M., DAVIS, J., FORBES, L., KOVACS, R., RUSSEL, W., SCHULTZ, L., SMITH, R., STANTON, J., and WAI, C.M. 1974. Heavy metal pollution in the sediments of the Coeur D'Alene River delta. July. Environ. Pollut. 7(1):1-6.

MAYACK, L.A., BUSH, P.B., FLETCHER, O.J., PAGE, R.K., and FENDLEY, T.T. 1981. Tissue residues of dietary cadmium in Wood Ducks. Arch. Environ. Contam. Toxicol. 10:637-645.

McKENNA, M.J., ZEMPEL, J.A., MADRID, E.O., and GEHRING, P.J. 1978. The pharmacokinetics of (^{14}C) vinylidene chloride in rats following inhalation exposure. Toxicol. Appl. Pharmacol. 45: 599-610.

MCLACHLAN, J.A., and DIXON, R.L. 1972. Gonadal function in mice exposed prenatally to p.p'-DDT. Toxicol. Appl. Pharmacol. 22:327.

MCLAUGHLIN, J. JR., MARLIAC, J.P., VERRETT, M.J., MUTCHLER, M.K., and FITZHUGH, O.G., 1964. Toxicity of 14 volatile chemicals as measured by the chick embryo method. Am. Ind. Hyg. Assoc. J.; 25:282-284.

MEISTER, R., (ed.) 1984. Farm chemicals handbook. 2nd ed. Willoughby, OH; Meister Publishing Co.

MENZIE, C.M. 1974. Metab Pesticides, p.154 (as cited in HSDB, 1988).

MESTITZOVA, M. 1967. On reproduction studies on the occurrence of cataracts in rats after long-term feeding of the insecticide heptachlor. Experientia 23:42-43.

MIETTINEN, J.K. 1973. Absorption and elimination of dietary mercury (Hg^{2+}) and methylmercury in man. In Miller, M., and Clarkson, T., (eds.) Mercury, Mercurials and Mercaptans. Charles C. Thomas, Springfield, IL.

MILLETTE, J.R., CLARK, P.J., STOBER, J., and ROSENTHAL, M. 1983. Asbestos in water supplies of the United States. Environ. Health Perspect., 53:45-48.

MITSUMORI, K., MAITA, K., TORU, S., SHUJI, T. and SHIRASU, U. 1981. Carcinogenicity of methylmercury chloride in ICR mice: preliminary note on renal carcinogenesis. Cancer Letters. 12:305-310. Elsevier/North-Holland Scientific Publishers Ltd.

MIZYUKOVA, I.G., and KURCHATOV, G.V. 1970. Metabolism of heptachlor. Farmacol. Toksiko. (Moscow) 33:499. CA73:86885j.

MOELLER, H.C., and RIDER, J.A. 1962. Plasma and red blood cell cholinesterase activity as indications of the threshold of incipient toxicity of ethyl-p-nitrophenol thionobenzenephosphorate (EPN) and malathion in human beings. Toxicol. Appl. Pharmacol., 4:123-136.

MONSANTO COMPANY. 1967. 13-Week Oral Administration—Dogs, Monochlorobenzene. U.S. EPA, OPTS, Washington, D.C. TSCA Sec 8(d) submission 8DHQ-1078-0202(2).

MOORE, J.W., and RAMAMOORTHY, S. 1984. Heavy Metals in Natural Waters. Springer-Verlag, New York.

MORIYA, M., OHTA, T., WATANABE, K., MIYAZAWA, T., KATO, K., and SHIRASU, Y. 1983. Further mutagenicity studies on pesticides in bacterial reversion assay systems. Mutat. Res. 116:185-216.

MRI (Midwest Research Institute). 1986. Subchronic (90-day) toxicity study of thallium sulfate in Sprague-Dawley rats. Office of Solid Waste, US EPA, Washington, D.C.

MUCKE, W., ALT, K.O., and ESSER, H.O. 1970. Degradation of 14C-labeled diazinon in the rat. J. Agr. Food Chem. 18:208-212.

MUNIR, K.M., SOMAN, C.S. and SAMATI, V. 1983. Hexachlorocyclohexane-induced tumorigenicity in mice under different experimental conditions. Tumori. 69:383-386.

MURATORE, F., FAIOLO, H., and PONZETTA, G. 1960. On four cases of poisoning by organophosphorus acid esters (antiparasitic) with recovery, with special reference to hepatic involvement. Minerva Med. 51:3342-3345.

NATIONAL ACADEMY OF SCIENCES (NAS). 1974. Vanadium. Committee of Biological Effects of Atmospheric Pollutants, Division of Medical Sciences, National Research Council. Washington, D.C.

NATIONAL ACADEMY OF SCIENCE (NAS). 1976. Health Effects of Benzene: A Review Committee on Toxicology, Assembly of Life Sciences. National Research Council, Washington, D.C.

NATIONAL ACADEMY OF SCIENCES. NAS. 1977. Safe Drinking Water Committee. Drinking Water and Health. Part 1, Chap. 1-5. Washington, D.C.: National Academy Press, pp.184-348.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980a. Recommended Dietary Allowances, 9th ed. Washington, D.C.

NATIONAL ACADEMY OF SCIENCES (NAS). 1980b. Mineral Tolerances of Domestic Animals. NAS, Washington, D.C.

NATIONAL CANCER INSTITUTE (NCI). 1976a. Report on the carcinogenesis bioassay of chloroform. Carcinogenesis Program, Division of Cancer Cause and Prevention, Washington, D.C.

NATIONAL CANCER INSTITUTE (NCI). 1976b. Carcinogenesis Bioassay of Trichloroethylene. CAS No. 79-01-6. Carcinogenesis Technical Report Series No. 2. PB-264-122.

NATIONAL CANCER INSTITUTE (NCI). 1976c. Report on Carcinogenesis Bioassay of Chloroform. N.C.I. Brief Communication.

NATIONAL CANCER INSTITUTE (NCI). 1977a. Bioassay of Tetrachloroethylene for Possible Carcinogenicity. CAS No. 127-18-4. NCI Carcinogenesis Technical Report Series No. 13. Washington, D.C. DHEW (NIH) Publication No. 77-813.

NATIONAL CANCER INSTITUTE (NCI). 1977b. Bioassay of Heptachlor for Possible Carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 9. DHEW Publication No. 77-809.

NATIONAL CANCER INSTITUTE (NCI). 1977c. Carcinogenesis Tech. Rep. Ser. No 14. Cas No 58-89-9. (As cited in Deichmann 1981).

NATIONAL CANCER INSTITUTE (NCI). 1977d. Bioassay of Chlordane for Possible Carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 8. 117 p. (Also publ. as DHEW Publication No. (NIH) 77-B08).

NATIONAL CANCER INSTITUTE (NCI). 1978a. Bioassay of Aldrin and Dieldrin for Possible Carcinogenicity. Bethesda. MD National Institute of Health. NCI-C6-TR-21. DHEW Publ. No. 78-821 184 pp.

NATIONAL CANCER INSTITUTE (NCI). 1978b. Bioasssays of DDT, TDE, and p,p'-DDE for Possible Carcinogenicity. NCI-CG-TR-1321.

NATIONAL CANCER INSTITUTE (NCI). 1978c. Bioassay of Endosulfan for Possible Carcinogenicity. Natl. Cancer Inst. Div. Cancer Cause and Prevention. Bethedsa, Maryland. DHEW Pub. No. (NIH) 78-1312 (As cited in EPA 1980b).

NATIONAL CANCER INSTITUTE (NCI). 1978d. Bioassay of Aroclor 1254 for Possible Carcinogenicity. Cas. No. 27323-18-8. NCI Carcinogenesis Technical Report Series No. 38. DHEW (NIH) Publication No. 78-838.

NATIONAL CANCER INSTITUTE (NCI). 1978e. Bioassay of Diazinon for Possible Carcinogenecity. Technical Report No. 137.

NATIONAL CANCER INSTITUTE (NCI). 1979a. Bioassay of Endrin for Possible Carcinogenicity. Technical Report. No. 12.

NATIONAL CANCER INSTITUTE (NCI). 1979b. Bioassay of Malathion for Possible Carcinogenicity. Technical Report. No. 24.

NATIONAL CANCER INSTITUE (NCI). 1979c. Bioassay of Malathion for Possible Carcinogenicity. Technical Report. No. 192.

NATIONAL CANCER INSTITUTE (NCI). 1979d. Bioassay of Diazinon for Possible Carcinogenicity. NCI Technical Report Series No. 137, Betheseda, MD.

NATIONAL CENTER FOR HEALTH STATISTICS CDC, USPHS. 1984. Blood lead levels for persons ages 6 months to 74 years: United States 1975-1980. (Data from the National Health and Nutrition Examination Survey). Vital Health and Statistics Series II, No. 233.

NATIONAL COFFEE ASSOCIATION (NCA). 1982. Twenty-four Month Chronic Toxicity and Oncogenicity Study of Methylene Chloride in Rats. Final Report. August 11, 1982. Prepared by Hazelton Laboratories America, Inc., Vienna, Virginia.

NATIONAL COFFEE ASSOCIATION (NCA). 1983. Twenty-Fourth-Month Oncogenicity Study of Methylene Chloride in Mice. Unpublished report prepared by Hazelton Laboratories, Inc., Vienna, Virginia.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1972. Occupational Exposure to Beryllium. HSM 72-10268. Washington, D.C.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1976. Criteria for a Recommended Standard—Occupational Exposure to Hydrogen Cyanide and Cyanide Salts (NsCN, KCN, and $Ca(CN)_2$). Washington, D.C. DHEW (NIOSH) Publication No. 77-108.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977a. Criteria for a Recommended Standard--Occupational Exposure to Coal Tar Products. DHEW (NIOSH) 78-107.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977b. Criteria for a Recommended Standard - Occupational Exposure to Vanadium. DHEW (NIOSH) Publication No. 77-222.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1977c. NIOSH Manual of analytical methods. 2nd ed. Vol. 2. Cincinnati, OH pp. 540-1-540-9.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1978. Special Occupational Hazard Review: Aldrin/Dieldrin. US DHEW Publ. No. 78-201.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1985a. Registry of Toxic Effects of Chemical Substances Data Base. Washington, D.C. July 1985.

NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH (NIOSH). 1985b. Pocket Guide to Chemical Hazards. USDHHS. p. 246-37.

NATIONAL RESEARCH COUNCIL (NRC). 1977a. Arsenic. National Academy of Sciences, Washington, D.C.

NATIONAL RESEARCH COUNCIL (NRC). 1977b. Drinking Water & Health, p. 540 (as cited in HSDB, 1988).

NATIONAL RESEARCH COUNCIL (NRC). 1980. Drinking Water and Health. Volume 3. Safe Drinking Water Committee. National Academy Press, Washington, D.C. 415 pp.

NATIONAL RESEARCH COUNCIL (NRC). 1982. An Assessment of Health Risks of Seven Pesticides Used for Termite Control. August 1982. NRC-P901. NTIS Publ. PB83-136374.

NATIONAL RESEARCH COUNCIL (NRC). 1988. Bier IV Report - Health Risks of Radon and Other Internally Deposited Alpha-Emitters. National Academy Press, Washington, D.C.

NATIONAL TOXICOLOGY PROGRAM/INTERAGENCY REGULATORY LIAISON GROUP (NTP/IRLG). 1982. The Conference on Phthalates. Environ. Health Perspect. 45:1-153.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1982a. Carcinogenesis Bioassay of Di(2-ethylhexyl) phthalate in F344 Rats and B6C3F_1 Mice. Feed Study. NTP Technical Report Series No. 217, U.S. Department of Health and Human Services. NIH Publication No. 82-1773. NTP-80-37.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1982b. Carcinogenesis bioassay of stanous chloride in F344/N rats and B6C3F1 mice (feed study). NCI/NTP Tech. Rep. Ser. No. 231.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1982c. Carcinogenesis bioassay of stannous chloride in F344 rats and B6C3F1 mice. Technical Report No. 231. Research Triangle Park. NC

NATIONAL TOXICOLOGY PROGRAM (NTP). 1983. Carcinogenesis Studies of Trichloroethylene (Without Epichlorohydrin), CAS No. 79-01-6, in F344/N rats and B6C3F_1 mice (Gavage Studies). Draft. August 1983. NTP 81-84, NTP TR 243.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1984a. Annual Plan for Fiscal Year 1984. Research Traingle Park, N.C. DHHS, Public Health Services. NTP-84-023 for 111, TCA.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1984b. Broad draft. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Chrysotile Asbestos in F344/N Rats. DHHS, Research Triangle Park, North Carolina. NTP TR 295.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986a. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Dichloromethane in F344/N Rats and B6C3F1 Mice (Inhalation Studies). NTP TR306.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986b. Toxicology and Carcinogenesis Studies of Tetrachloroethylene (Perchloroethylene) CAS No. 127-18-4) in F344/N Rats and B6C3F1 Mice (Inhalation Studies). NTP Technical Report Series No. 311. Research Triangle Park, North Carolina. DHEW (NIH) Publication No. 86-2567.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986c. Toxicology and Carcinogenesis Studies of xylenes (mixed) [60% m-xylene; 14% p-xylene, 9% O-xylene; 17% ethylbenzene] in F344/N rats and B63F1 mice (Gavage studies) December 1986. NTP TR 327.

NATIONAL TOXICOLOGY PROGRAM (NTP). 1986d. Toxicology and Carcinogenesis Bioassay of Benzene in F344/N Rats and B6CF1 Mice. Gavage Studies. NTP Technical Report Series #289. Research Triangle Park, NC

NAWROT, P.S., and STAPLES, R.E. 1979. Embryo-fetal toxicity and teratogenicity of benzene and toluene in the mouse. Teratology 19:41A.

NEAL, J., and RIGDON, R.H. 1967. Gastric tumors in mice fed benzo(a)pyrene: A quantitative study. Tex. Rep. Biol. Med. 25:553-557.

NIKONOROW, M., et al. 1973. Effect of orally administered plasticizers and polyvinyl chloride stabilizers in the rat. Toxicol. Appl. Pharmacol. 26:253.

NORBACK, D.H., and WELTMAN, R.H. 1985. Polychlorinated biphenyl induction ofhepatocellular carcinoma in the Sprague-Dawley rat. Environ. Health Perspect. 1:134-143.

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA). 1986. 20 CFR Parts 1910 and 1926. Occupational exposure to asbestos, tremolite, anthophyllite, and actinolite. June 20, 1986. Fed. Reg. 51:22612-22790.

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION (OSHA). 1989. 29 CFR Part 1910.1000. Air Contaminants-Permissible Exposure Limits. July, 1989.

O'SHEA, T.A., AND MANCY, K.H. 1978. The effect of pH and hardness on the competitive interaction between trace metal ions and inorganic and organic complexing agents found in natural waters. Water Res. 12;703-711.

ONG, H.L., SWANSON, V.E., and BISQUE, R.E. 1970. Natural organic acids as agents of chemical weathering. U.S. Geological Survey Professional Paper 700-C:C130-137.

OTT, M.G., TOWNSEND, J.C., FISHBECK, W.A., and LANGNER, R.A. 1978. Mortality among individuals occupationally exposed to benzene. Arch. Environ. Health 33:3-10.

OTT, W.R. 1988. A physical explanation of the lognormality of pollution concentrations. Presented at the 81st Annual Meeting of the Air Pollution and Hazardous Waste Management Association. June 19-24, 1988. Dallas, TX.

OTTOLENGHI, A.D., HASEMAN, J.K., and SUGGS, F. 1974. Teratogenic effects of aldrin, dieldrin, and endrin in hamsters and mice. Teratology 9:11-16 (As cited in EPA 1985g).

PALMER, A.K., BOTTOMLEY, A.M., WARDEN, A., FROHBERG, H., and BAUER, A. 1978. Toxicology 9(3), 239. (As cited in Deichmann 1981).

PARRIS, G.E., and BRINKMAN, F.E. 1976. Reactions which relate to the environmental mobility of arsenic and antimony. II. Oxidation of trimethylarsine and trimethylstibine. Environ. Sci. Technol. 10(12):1128-1134.

PATTEE, O.H. 1984. Eggshell thickness and reproduction in American Kestrals exposed to chronic dietary lead. Arch. Environ. Contam. Toxicol. 13:29-34.

PEARSON, C.R., and MCCONNELL, G. 1975. Chlorinated C_1 and C_2 hydrocarbons in the marine environment. Prog. R. Soc. Lond. B. 189:305:332 (As cited in ATSDR 1987).

PERRY, H.M., KOPP, S.J., ERLANGER, M.W., and PERRY, E.G. 1983. XVII. Cardiovascular effects of chronic barium ingestion. In Hemphill, D.D., (ed.) Proceedings of the University of Missouri's 17th Annual Conference of Trace Substances in Environmental Health. University of Missouri Press, Columbia, Missouri (As cited in EPA 1984).

PLANNING RESEARCH CORPORATION (PRC). 1985. The Endangerment Assessment Handbook. August. Prepared for U.S. EPA, Office of Waste Programs Enforcement.

POHLMAN, A.A., and McCALL, J.G. 1986. Kinetics of metal dissolution from forest soil by soluble organic acids. J. Environ Quality 15:86-92.

PORIES, W.J., HENZEL, J.H., ROB. C.G., and STRAIN, W.H. 1967. Acceleration of wound healing in man with zinc sulfate given by mouth. Lancet. 1: 121-124.

PORTER, K.G., MCMASTA, D., ELMES, M.E., and LOVE, A.G. 1977. Anemia and low scrum-copper during zinc therapy. Lancet, Vol. 8:744.

POURBAIX, M. 1963. Atlas d'Equilibres Electrochemiques. Gauthier-Villar and Co., Paris.

PRASAD, A.S., SCHOOMAKER, E.B., ORTEGA, J., et al. 1975. Zinc defiency in sickle cell disease. Clin. Chem. 21:582-587.

PROBST, G.S., McMAHON, K.E., HILL, L.E., et al. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 compounds. Environ. Mutagen. 3:11-32 (As cited in EPA 1985).

QUAST, J.F., HUMISTON, C.G., WADE, C.E., BALLARD, J., BEYER, J.E., SCHWETZ, R.W., and NORRIS, J.M. 1983. A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidene chloride. Fund. Appl. Toxicol. 3: 55-62.

RADDING, S.B., LIU, D.J., JOHNSON, H.L., and MILL, T. 1976. The Environmental Fate of Selected Polynuclear Aromatic Hydrocarbons. EPA 560/5-75-009.

RALEIGH, R.L. and MCGEE, D. 1972. Effects of short high concentration exposures to acetone as determined by observation in the work areas. J. Occup. Med. 14:607-610.

RASHID, M.A, and LEONARD, J.D. 1973. Modifications in the solubility and precipitation behavior of various metals as a result of their interactions with sedimentary humic acid. Chem. Geol. 11: 89-97.

RAMAMOORTHY, S., and RUST, B.R. 1978. Heavy metal exchange processes in sediment-water systems. Environ. Geol. 2:165-172.

RAY-BETTLEY, F., and O'SHEA, J.A. 1975. The absorption of arsenic and its relation to carcinoma. Br. J. Dermatol. 96:563 (as cited in EPA, 1980).

REUBER, M.D. 1978. Carcinomas, sarcomas and other lesions in Osborne-Mendel rats ingesting endrin. Exp. Cell. Biol. 46:129-145.

REUBER, M.D. 1979. Carcinogenicity of endrin. Sci. Total Environ. 12:101-135.

REXRODE, M. 1987. Ecotoxcity of tributyltin. in: The Ocean, an International Workplace. Proceedings. Volume Four, International Organotin Symposium. Marine Technology Society. September 28 - October 1, 1987. Halifax, Nova Scotia, Canada. Pp. 1443-1455.

RINSKY, R.A., YOUNG, R.J., and SMITH, A.B. 1981. Leukemia in benzene workers. Am. J. Ind. Med. 3:217-245.

ROSSI, L., RAVERA, M., REPETTI, G., and SANTI., L. 1977. Long-term administration of DDT or phenobarbital-NA in Wistar rats. Int. J. Cancer, 19:179-185 (as cited in EPA, 1988a).

RUMACK, B.H. (ed.) 1986. Poisindex. Microfiche ed. Micromedix, Inc., Denver, Colorado, in association with the National Center for Poison Information, with updates, 1975-present.

RUSSELL, D.J. and MCDUFFIE, B. 1986. Chemodynamic properties of phthalate esters: Partitioning and soil migration. Chemosphere. 15:1003-1021.

SANTODONATO, J., HOWARD, P., and BASU, D. 1981. Health and ecological assessment of polynuclear aromatic hydrocarbons. J. Envrion. Pathol. Toxicol. 5:1-364.

SCHAEFFER, E., GREIM, H., and GOESSNER, W. 1984. Pathology of chronic polychlorinated biphenyl (PCB) feeding in rats. Toxicol. Appl. Pharmacol. 75:278-288.

SCHMAL, D. 1955. Testing of Naphthalene and Anthracene as Carcinogenic Agents in the Rat. Z. Krebsforsch. 60:697-710 (German with English translation).

SCHMAL, D., and STEINHOFF, D. 1960. Versuche zur Krebserzeugung mit kolloidalen Silber- und Goldlosungen an Ratten. Z. Krebsforsch. 63:586-591 (As cited in EPA 1985y).

SCHMIDT, R. 1973. Effects of 1,1,1-trichloro-2,2-bis(p-chlorophenol)-ethane (DDT) on the prenatal development of the mouse. Biol. Rundsch. 11:316-317 (Ger.).

SCHNEIDERMAN, M., NISBET, I.C.T., and BRETT, S.M. 1981. Assessment of risks posed by exposure to low levels of asbestos in the general environment Bga - Berichte 4:31-37.

SCHROEDER, H.A., MITCHNER, M., and NASAR, A.P. 1970. Zirconium, niobium, antimony, vanadium, and lead in rats: Lifetime studies. J. Nutr. 100:59-66.

SCHROEDER, H.A., and MITCHENER, M. 1975a. Life-term effects of mercury, methylmercury and nine other trace metals on mice. J. Nutr. 105:452-458.

SCHROEDER, H.A., and MITCHNER, M. 1975b. Life-term studies in rats: Effects of aluminum, barium, beryllium, and tungsten. J. Nutr. 105:421-421. (As cited in EPA 1984).

SCHWETZ, B.A., LEONG, B.K.J., and GEHRING, P.J. 1974. Embryo- and fetotoxicity of inhaled carbon tetrachloride, 1,1-dichloroethane and methyl ethyl ketone in rats. Toxicol. Appl. Pharmacol. 28:452-464.

SCHWETZ, B.A., LEONG, B.K.J., and GEHRING, P.J. 1975. The effect of maternally inhaled trichloroethylene, perchloroethylene, methyl chloroform, and methylene chloride on embryonal and fetal development in mice and rats. Toxicol. Appl. Pharmacol. 55:207-219.

SEIDLER, H., MACHOLZ, R.M., HAERTIG, M., KUJAWA, M. and ENGST, R. 1975. Nahrung, 19(5/6), 473. (As cited in Deichmann 1981).

SEILER, J.P. 1979. Herbicidal phenylalkylurcas as possible mutagens. II. Chemical basis of mutagenic activity. Pestic. Biochem. Physiol. 12(2) 183.

SESHAN, K. 1983. How are the physical and chemical properties of chrysotile asbestos altered by a 10-year residence in water and up to 5 Days in simulated stomach acid? Environ. Health Perspec., 53: 143-148.

SHEPARD, T.A. 1986. Catalog of Teratogenic Agents, 5th Ed., Johns Hopkins University Press, Baltimore and London, p. 186.

SHERMAN and CULIK, 1971. Report on Monuron, Haskell Lab. Report No. 300-17. EPA Pesticide petition No. 17, Vol. I.

SHERMAN H., and KAPLAN A.M., 1975. Toxicity studies with 5-bromo-3-sec-butyl-6-methyluracil. Toxicol Appl Pharmacol 34 (Nov): 189-96 (1975).

SIMMON V.F. et al. 1976. In vitro mutagenic studies of twenty pesticides. Toxicol Appl Pharmacol 37 (1): 109 (1976).

SNYDER, C.A., GOLDSTEIN, B.D., SELLAKUMAR, A.R., BROMBERG, I., LASKIN, S., and ALBERT, R.E. 1980. The inhalation toxicology of benzene: Incidence of hematopoietic neoplasms and hematotoxicity in AKR/J and C57BL/6J mice. Toxicol Appl Pharm. p. 361.

SPENCER, 1982. Guide to Chem in Crop Protect, p. 406 (as cited in HSDB, 1988).

SPYKER, J.M., and AVERY, K.L. 1977. Neurobehavioral effects of prenatal exposure to the organophosphate diazinon in mice. J. Toxicol. Environ. Health 3:989-1002.

STEINBERG, C. 1980. Species of dissolved metals derived from oligotrophic hard water. Water Research. 14: 1239-1250.

STEWART, R.D., HAKE, C.L., FORSTER, H.V., LEBRUN, A.J., PETERSON, J.F., and WU, A. 1974. Tetrachloroethylene: Development of a biologic standard for the industrial worker by breath analysis. Report No. NIOSH-MCOW-ENUM-PCE-74-6, Medical College of Wisconsin, Milwaukee, Wisc.

STOKINGER, H.E., 1981. The Metals. In: Patty's Industrial Hygiene and Toxicology, 3rd Ed., Vol. IIA. Clayton, G.D., and Clayton, F.E. (eds.), John Wiley and Sons, New York. pp. 1493-2060.

TABAK, H.H., QUAVE, S.A., MASHNI, C.I., and BARTH, E.F. 1981. Biodegradability studies with organic priority pollutant compounds. Journal WPCF. 53:1503-1518.

TAI, C.N., BRECKENRIDGE, C., and GREEN, J.D. 1985a. Simazine technical subacute oral 13-week toxicity study in rats. Ciba-Geigy Pharmaceuticals Division. Report No. 85018, Acc. No. 257693.

TAI, C.N., BRECKENRIDGE, C., and GREEN, J.D. 1985b. Simazine technical subacute oral 13-week toxicity study in dogs. Ciba-Geigy Pharmaceuticals Division. Report No. 85018, Acc. No. 257692.

TAKEUCHI, Y., ONO, Y., HISANAGA, N., et al. 1983. An experimental study of the combined effects of n-hexane and methyl ethyl ketone. Br. J. Ind. Med. 40:199-203.

TARASENKO, M., PROMIN, O., and SILAYEV, A. 1977. Barium compounds as industrial poisons (an experimental study). J. Hyg. Epiderm. Microbial. Immunol. 21:361 (As cited in EPA 1984).

TELAND, S., TONG, C., and WILLIAMS, G.M. 1981. Induction of mutagenesis by carcinogenic polycyclic aromatic hydrocarbons but not by organochlorine pesticides in the ARL/HGPRT mutagenesis assay. 12th Ann. Meet. Environ. Mutagen Soc. Environ. Mutagen. 3:359.

TERRACINI, B., TESTA, M.C., CABRAL, J.R.P., and DAY, N. 1973. The effects of long-term feeding of DDT to BALB/c mice. Int. J. Cancer. 11:747-764.

THORPE, E. and WALKER, A.I.T. 1973. Toxicology of dieldrin (HEOD). II. Comparative long-term oral toxicity studies in mice with dieldrin, DDT, phenobarbitone, beta-HCH and gamma-HCH. Food Cosmet. Toxicol. 11:433-442.

THUN, M.J., SCHNORR, T.M., SMITH, A.B., HALPERIN, W.E., and LEMEN, B.A. 1985. Mortality among a cohort of U.S. Cadmium production workers - An update. JNCI 74:325-333.

THYSSEN, J., ALTHOFF, J., KIMMERLE, G., and MOHR, U. 1981. Inhalation studies with benzo(a)pyrene in Syrian golden hamsters. J. Natl. Cancer Int. 66:575-577.

TOMATIS, L., TURUSOV, V., CHARLES, R.T., BIOCCHI, M., and GATI, E. 1974. Liver tumors in CF-1 mice exposed for limited periods to technical DDT. Z. Krebsforsch. 82:25-35.

TOMATIS, L., and TURUSOV, V. 1975. Studies in the carcinogenicity of DDT. Gann. 17: 219-241.

TORKELSON, T.R., OYEN, F., MCCOLLISTER, D.D., and ROWE, V.K. 1958. Toxicity of 1,1,1-Trichloroethane as determined in laboratory animals and human subjects. Am. Ind. Hyg. Assoc. J. 19:353-362.

TORKELSON, T.R., and ROWE, V.K. 1981. Halogentated aliphatic hydrocarbons containing chlorine, bromine and iodine. In: Patty's Industrial Hygiene and Toxicology, Vol. IIb, 3rd Revised Edition. G.D. Clayton and F.E. Clayton, Eds. John Wiley and Sons, NY, pp. 3443-3602.

TREON, J., and CLEVELAND, F.P. 1955a. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals with special reference to dedrin/dieldrin. Agri.food. Chem. Journ. 3:402.

TREON, J.F., CLEVELAND, F.P., and CAPPEL, J. 1955b. Toxicity of endrin for laboratory animals. J. Agric. Food Chem. 3:842-848.

TSENG, W.P., CHU, H.M., HOW, S.W., FONG, J.M., LIN, C.S., and YEH, S. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40:453-463.

TURUSOV, V.S., DAY, N.E., TOMATIS, L., GATI, E., and CHARES, R.T. 1973. Tumors in CF-1 mice exposed for six consecutive generations to DDT. J. Natl. Cancer Inst. 51: 983-998.

U.S. ARMY CORPS OF ENGINEERS. 1988. Oakland Harbor, Deep-draft Navigation Improvements Design Memorandum Number 1, General Design and Final Supplement 1 to the Environmental Impact Statement Alameda County California.

U.S. DEPARTMENT OF AGRICULTURE (USDA). 1984. United States Department of Agriculture. Forest Service. Pesticide background statements. Vol. 1. Herbicides.

U.S. DEPARTMENT OF AGRICULTURE (USDA). 1987. New England Wildlife and Habitat, Natural History, and Distribution. General Technical Report NE-108.

U.S. DEPARTMENT OF AGRICULTURE (USDA). 1988. Draft Environmental Impact Statement. Vegetation Management in the Coastal Plain/Piedmont. Appendices. Volume II appendices. Prepared by Labatt-Anderson, Inc. Arlington, VA

U.S. DEPARTMENT OF HEALTH, EDUCATION & WELFARE (USDHEW). 1969. Report of the Secretary's Commission on Pesticides and Their Relationship to Environmental Health.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES (USDHHS). 1982. National Toxicology Program Annual Plan Fiscal Year 1982. NTP-81-94.

URBAN, D.J. and COOK, N.J. 1986. Standard Evaluation Procedures for Ecological Risk Assessment. Hazard Evaluation Division. Environmental Protection Agency. Washington, D.C.

UZHDOVINI, E.R., et al. 1974. Acute toxicity of lower phenols. Gig. Tr. Prof. Zabol. 2:58.

VAN OETTINGEN, W.F., NEAL, P.A., DONOHUE, D.D., et al. 1942a. The toxicity of potential dangers of toluene, with special reference to its maximal permissible concentration. U.S. Public Health Service Pub., Bull. No. 279. p. 50 (as cited in EPA 19871).

VAN RAALTE, HGS. 1977. Human experience with dieldrin in perspective. Ecotoxicol. Environ. Saf. 1:203-210.

VELSICOL CHEMICAL CORPORATION. 1973. Available from EPA. Write to FOI, EPA Washington, D.C., 20460 (as cited in EPA 1988a).

VENUGOPAL, B. and LUCKEY, T.D. 1978. Metal toxicity in mammals. In: Chemical Toxicity of Metals and Metalloids. Plenum Press, New York.

VERSAR, INC. 1975. Preliminary Investigation of Effects on the Environment of Boron, Indium Nickel, Selenium, Tin, Vanadium and Their Compounds. Prepared for the EPA. Vo. V. Tin. PN-245-988.

VERSCHUEREN, K. 1983. Handbook of Environmental Data on Organic Chemicals. 2nd ed. Van Nostrand Reinhold Co., New York.

VINOGRADOV, A.P. 1959. The Geochemistry of Rare and Dispersed Chemical Elements in Soil. 2nd ed. New York: Consultants Bureau, Inc. Cited in 1985.

VOGEL, T.M., CRIDDLE, C.S., and MCCARTY, P.L. 1987. Transformations of halogenated aliphatic compounds. Environ. Science and Technology; 21(8):722-736.

VON OETTINGEN, W.F., NEAL, P.A., DONAHUE, D.D., et al. 1942b. The toxicity and potential dangers of toluene – Preliminary Report. J. Am. Med. Assoc. 118:579-584 (As cited in EPA 1987h).

WADE, T.L., GARCIA-ROMERO, B., and BROOKS, J.M. 1988. Tribulytin analyses in association with NOAA's national status and trends mussel watch program. in: Oceans '88, A Partnership of Marine Interests. Proceedings. Volume 4. October 31 - November 9, 1988. Marine Technology Society, Baltimore, Maryland. Pp. 1198-1201.

WAGONER, J.K., INFANTE, P.F., and BAYLISS, D.L. 1980. Beryllium: An etiologic agent in the induction of lung cancer, neoplastic respiratory disease, and heart disease among industrially exposed workers. Environ. Res. 21:15-34. (As cited in EPA 1986c).

WAHLER ASSOCIATES, 1985 (Wahler/WESTDIV). Draft Report - Verification Step Confirmation Step Confirmation Study, Draft Report of Navy Contract No. N62474-83-C-3075, Report to Commanding Officer, Western Division, Naval Facilities Engineering Command, San Bruno, California.

WALKER, A.I.T., STEVENSON, D.E., ROBINSON, J., THORPE, E., and ROBERTS, M. 1969. The toxicology and pharmacodynamics of dieldrin (HEOD): Two-Year oral exposure of rats and dogs. Toxicol Appl. Pharmacol. 15:345-373.

WALKER, W.W., and STOJANOVIC, B.J. 1973. J Environ Qual 2: 229-32 (as cited in HSDB, 1988).

WALSH, A.H., and RIBELIN, W.E. 1973. The Pathology of Fish. Ribelin, W.E., Migaki, G., eds., U of Wisconsin Press, Madison WI pp. 515-H. (as cited in HSDB, 1988).

WANG, H.H., and MCMAHON, B. 1979. Mortality of workers employed in the manufacture of chlordane and heptachlor. J. Occup. Med. 21:745-748.

WEAST, R.E., ed. 1983. Handbook of Chemistry and Physics. 63rd ed. CRC Press, Cleveland, OH.

WEDEN, G.P., et al. 1984. Renal involvement in organophosphate poisoning. J. Am. Med. Assoc. 252:1408.

WELTY, J.C. 1982. The Life of Birds. 3rd Edition. Saunders College Publishing.

WHITE, D.H. and FINLEY, M.T. 1978. Uptake and retention of dietary cadmium in Mallard ducks. Environ. Res. 17:53-59.

WHITE, D.H., FINLEY, M.T., and FERRELL, J.F. 1978. Histopathologic effects of dietary cadmium on kidneys and testes of mallard ducks. J. Tox. Environ. Health. 4:551-558.

WITHERUP, S., STEMMER, K.L., TAYLOR, P., and BIETSCH, P. 1970. The Incidence of Neoplasms in Two Strains of Mice Sustained on Diets Containing Endrin. Kettering Lab., Univ. Cincinnati, Cincinnati, OH

WHITE-STEVENS. 1976. Pest In Environ. Vol. 2, p. 319 (as cited in HSDB, 1988).

WILLIAMS, G.M. 1980. Classification of genotoxic and epigenetic hepatocarcinogens using liver culture assays. Ann. N.Y. Acad. Sci. 349:273-202.

WILSON, B.H., SMITH, G.B., and REES, J.F., 1986. Biotransformations of selected alkyl benzenes and halogenated aliphatic hydrocarbons in methanogenic aquifer material: A microcosm study. Environ. Sci. Technol., 20(10):997-1002.

WOLF, M.A., ROWE, V.K., McCOLLISTER, D.D., HOLLINGSWORTH, R.C., and OYEN, F. 1956. Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health 14:387-398.

WONG, O., MORGAN, R.W., and WHORTON, M.D. 1983. Comments on the NIOSH Study of Leukemia in Benzene Workers. Technical report submitted to Gulf Canada, Ltd. by Environmental Health Associates.

WOODARD, G., WOODARD, M., and CRONIN, M.T.I. 1968. Safety evaluation of the pesticide diazinon by a two-year feeding trial in rhesus monkeys. Fed. Proc., 27:597.

WOODARD RESEARCH CORPORATION (WRC). 1965. Three-generation reproduction study of simazine in the diet of rats. MRID 00023365, 00080631.

WORLD HEALTH ORGANIZATION (WHO). 1976. Environmental Health Criteria, Mercury. Geneva 12dichl.

WORLD HEALTH ORGANIZATION (WHO). 1984. Endosulfan. Environmental Health Criteria 40. World Health Organization. Geneva.

WORTHING. 1983. The Pesticide Manual. British Crop Protection Council. 7th. ed. Nottingham, England. p. 228 (as cited in HSDB 1988).

WORTHING, C.R., WALKER, S.B. 1987. The Pesticide Manual. British Crop Protection Council. 8th ed. Nottingham, England. 449-450.

WORTHING, P. 1988. Personal communication with Patty Worthing. October 1988.

WYLIE. A.G., VIRTA, R.L., and SEGRETTI, J.M. 1988. Characterization of mineral population by index particle: Implication for the Stanton hypothesis. Environ. Res. 43:427-439.

YANG, G., WANG, S., ZHOU, R., and SUN, S. 1983. Endemic selenium intoxication of humans in China. Am. J. clin. Nutr. 37:872-881.

YELIZAROV, G.P. 1977. Occupational skin disease caused by simazine and propazine. Pest. Abstr. 6:73-0352.

ZAPP, J.A., Jr. 1955. Report on Monvron, EPA Pesticide Petition No. 17, Vol.II.

ZOECON CORPORATION. 1983. MRID No. 00128356. As cited in EPA 1988a. Available from EPA.

APPENDIX A

CHEMICAL ANALYSIS OF GROUNDWATER FROM THE PAN AMERICAN WELL AT NAS ALAMEDA

TABLE A-1

Major Inorganic and Trace Mineral
Analyses for Pan American Well

Date Collected	6/12/77
Pumping Rate	398.3 gpm
Ph, units	7.40
Temperature, °C	20.4
Electrical Conductivity, μmhos/cm @ 25°C	890
TDS (sum), mg/l	588.62
Color (Pt-Co Unit)	.5
Odor (threshold unit)	1

(All values in mg/l, unless otherwise stated.)

HCO_3-	240
Cl-	110.5
SO_4=	33.4
F-	.4
NO_3- (N)	.1
NO_2- (N)	.002
Na+	120
K+	1.9
Ca++	36.9
Mg++	14.6
SiO_2	30.3
As	< 0.01
Ag	< 0.008
Ba	0.19
B	< 0.1
Cd	0.005
Cr	< 0.001
Cu	< 0.01
Cn	< 0.01
Fe	< 0.01
Hg	0.011
Mn	0.07
Pb	< 0.05
Se	< 0.001
Zn	0.075
Hardness (as $CaCO_3$)	92.0

Analysis Agency: International Nutronics, Inc., Palo Alto, California 94303
Taken from Hydro-Search/Navy Public Works, 1977

APPENDIX B

ANIMAL SPECIES OF NAS ALAMEDA AND THE ADJACENT WATERS

TABLE B-1

BIRDS OF THE NAS ALAMEDA REGION

Common Name	Scientific Name
Eared grebe	Podiceps caspicus
Red-throated loon	Gavia stellata
Piedbilled grebe	Podilymbus podiceps
Western grebe	Aechmophorus occidentalis
Doublecrested cormorant	Phalacrocorax auritus
Great blue heron	Ardea herodias
Common or American egret	Casmerodius albus
Snowy egret	Leucophoyx thula
Canvasback	Aythya valisineria
Lesser scaup	Authya affinis
Greater scaup	Aythya marila
Common goldeneye	Bucephala clanqula
Bufflehead	Bucephala albeola
White-winged scoter	Melanitta deglandi
Surf scoter	Melanitta perspicillata
Ruddy duck	Oxyura jamaicensis
Brown pelican	Pelecanus occidentalis
White pelican	Pelecanus erythrorhynehos
American coot	Fulica americana
Killdeer	Charadrius vociferus
Semipalmated plover	Charadrius semipalmatus
Snowy plover	Charadrius alexandrinus
Common snipe	Capella gallinago
Black-bellied plover	Squatarola squaiarola
Long-billed curlew	Numenius americanus
Hudsonian curlew	Numenius phacopus
Willet	Catoptrophorus semipalmatus
Least sandpiper	Erolia minutilla
Dunlin	Erolia alpina
Western sandpiper	Ereunetes mauri
Short-billed dowitcher	Limnodromus griseus
Marbled godwit	Limosa fedoa
Black turnstone	Arenaria melanocephala
Avocet	Recurvirostra americana
Black-necked stilt	Himantopus mexicanus
Northern phalarope	Lobipes lobatus
Western gull	Larus occidentalis
Herring gull	Larus argentatus
California gull	Larus californicus

TABLE B-1 (Continued)

BIRDS OF THE NAS ALAMEDA REGION

Common Name	Scientific Name
California least tern	Sterna anillarum browni
Ring-billed gull	Larus delawarenis
Bonaparte's gull	Larus philadelphia
Glausous-winged gull	Larus glaucescens
Forster tern	Sterna forsteri
Caspian tern	Hydroprogne caspia
Loggerhead shrike	Lanius ludovicicanus
Black-shouldered kite	Elanus caeruleus
Red-tailed hawk	Buteo jamaicensis
Northern harrier	Circus cyaneus
American kestrel	Falco sparverius
Peregrin falcon	Falco peregrinus
Burrowing owl	Athene cunicularia

Source: Environmental Assessment Engineering, June 1974, Preliminary Submission Candidate Environmental Impact Statement for Dredge Soil Operations at the Naval Air Station, Alameda, California (As cited in E&E/WESTDIV, 1983)

TABLE B-2

MAMMALS OF NAS ALAMEDA

Common Name	Scientific Name
Black-tailed jackrabbit	Lepus californicus
Vole	Microtus sp.
House mouse	Mus musculus
Western harvest mouse	Reithrodontomys megalotis
Western ground squirrel	Citellus beecheyi
Raccoon	Procyon loter
Feral cat	Felis domesticus

TABLE B-3

AQUATIC INVERTEBRATE SPECIES OF WATERS ADJACENT TO NAS ALAMEDA

Common Name	Scientific Name
Polychaete worms	Asychis amphiglypta
	Cirriformia spirabrancha
	Cirratulus cirratus
	Dorvillea gracilis
	Glycinde armigera
	Haploscoloplos elongata
	Naphtys caecoides
	Streblospio benedicti
	Sabellidae
White acorn barnacle	Balanus glandus
Mud crab	Hemigrapsus oregonesis
	Rithropanopeus harrissi
Mud snail	Nassarius mendicus
Bay mussel	Mytilus edulis
California mussel	Mytilus californicus
Native oyster	Ostrea lurida
Littleneck clam	Porthothaca staminea
Japanese littleneck clam	Tapes semidecussata
Soft-shelled clam	Mya arenaria
Bent-nosed clam	Macoma nasuta

Source: Environmental Assessment Engineering, June 1974, Preliminary Submission Candidate Environmental Impact Statement for Dredge Soil Operations at the Naval Air Station, Alameda, California (As cited in E & E/WESTDIV, 1983)

TABLE B-4

FISH OF THE NAS ALAMEDA REGION

Common Name	Scientific Name
Pacific herring	Clupea harengus
Northern anchovy	Engraulis mordax
Jack smelt	Atherinopsis californiensis
Top smelt	A. affinis
Starry flounder	Platichthys stellatus
Shad	Dorosoma sp.
Surfperch	Hyperprosopon agenteum
Striped bass	Morone saxatilis
Chinook salmon	Oncorhynchus tshawytscha
Coho salmon	O. kisutch
White croaker	Genyonemus linneatus
Yellowfin goby	Acanthogobius flavinanus